THE
LARYNGOSCOPE.

VOL. XLIX AUGUST, 1939. No. 8

RHINOLOGY IN CHILDREN. RESUME OF AND COMMENTS ON THE LITERATURE FOR 1938.

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[Systematic search of 10 prominent journals has revealed very few articles exclusively devoted to rhinology in children. A number of those disclosed sound work, intelligent planning and concise, clear presentation; the originals deserve study. Certain other articles on various aspects of rhinology have parts referring to children; the pertinent portions of these are indicated in the following abstracts. In addition, mention is made of certain articles which touch on problems of diagnosis or treatment in children.—ED.]

Two concisely written descriptions of investigations carefully planned to show the characteristics of sinusitis in children appear in British journals.

Ebbs[1] examined the sinuses of children postmortem. In 496 children, 152 were found to have purulent infection in one or more sinuses. In other words, 30.6 per cent of the children of all ages who died in a large children's hospital showed evidence of sinusitis at necropsy.

Infection was recorded as present only when definite pus or mucopus was found in the sinus; sinuses that contained thin watery or clear mucus were considered normal.

Thirty-two and six-tenths per cent of 331 infants under 12 months of age, and 33.3 per cent of 60 children in the second year of life had infected sinuses. Of 10 infants 1 month or under who had pus in the antra, two were only 2 weeks old

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, July 31, 1939.

at death. (Infection of the antrum in an infant only 3 days old was recorded by Holt and McIntosh, 1933.)

The view that some patients develop a collection of fluid in the sinuses during the terminal period of a fatal illness, when resistance and normal ciliary action are poor, cannot be satisfactorily disproved in all cases, but the history and clinical course, in addition to the gross sepsis found at necropsy, in the majority of these infants and children, is very strong evidence in favor of a preterminal infection. The incidence of the common cold and frequency of respiratory infections in the patients who are brought to a children's hospital are high enough to support the view that a considerable number have an infection in the accessory nasal sinuses.

The part which sinusitis has played as a cause of death in this group of children was not assessed since it could have been the etiological factor or an associated condition or the result of the condition which caused the death.

Bowen-Davies[2] investigated the sinuses of child patients. He examined the maxillary antra in 55 children between the ages of 5 and 14 years.

In each case a routine investigation was carried out. An X-ray photograph was taken, the nose was cocainized and swabbed, and both antra were punctured, making a total of 110 antrum punctures. The washings from the antra were cultured and the antra filled with lipiodol. Further, X-ray photographs were taken immediately and at intervals of a week or so, until the antra were clear of lipiodol. The volume of lipiodol injected and the time it took to disappear were recorded. In the early cases, photographs were taken every 24 hours, and it soon became apparent that not only was the cost of the investigation and inconvenience to the patient too great but the safe dose of X-rays would be exceeded before the antra were empty. By observing the progress of the lipiodol it was possible to place the X-ray examination in such a way that the final examination coincided with the complete evacuation of the lipiodol.

For antrum puncture, local anesthesia was used with ease in all except four cases. An attempt was made to aspirate the contents of the antrum with a dry, sterilized glass syringe. If the antrum appeared empty, 2 cc. of sterile saline were

injected and aspirated. The specimen was immediately centrifuged and planted on blood agar. In the early cases, lipiodol was injected until the antrum was full. The capacity of some of the antra was found to be 8 cc. or more, and it was decided to limit the injections to 5 cc. This did not apply in the majority of the infected cases since the capacity of the antrum was less than 5 cc.

Of the 55 cases investigated, organisms were found in the antra of 23. Bilateral infection occurred in 12 patients, and of 110 antra, 35 were infected. In 19 patients, mucopus was found in one or both antra. It was present in both sides in four patients, and out of 110 antra, mucopus was, therefore, present in 23. In six of these, cultures proved to be sterile; infected mucopus was, therefore, present in 17 antra. Infection without mucopus occurred in 12 antra.

The author gives five tables presenting the statistics regarding the organisms found.

The X-ray photographs were all taken with the patient in the supine position. Of 110 antra, 66 appeared to be infected on X-ray examination. Of these 66, 27 subsequently proved to be infected, and 39 proved sterile. Of the 44 cases with a negative skiagram, 35 proved to be sterile, and nine to be infected. Of the 23 antra containing mucopus, 17 appeared infected on X-ray examination, six appeared normal. It has been Bowen-Davies' experience on several occasions to see a negative X-ray photograph in a case of acute frontal sinusitis in which the sinus was subsequently found full of pus. The skiagram may not demonstrate the presence of mucopus. It will usually show a thickened membrane. It has never been able to demonstrate an organism, and it is in the deduction that a thickened membrane indicates infection that we are wrong. The pathology of a thickened membrane without infection is obscure, but sometimes the condition may be of an allergic nature.

The average rate of drainage in sterile cases was 4.9 days per cubic centimetre of lipiodol. The average time for a complete evacuation was 17.9 days. This figure makes no allowance for the fact that many of the antra were not completely filled.

He discusses the infected antrum which did not contain mucopus. It has been suggested that such an antrum is infected but is draining adequately. In this series, all the infected antra drained as rapidly as the sterile antra, and in this series all the antra containing mucopus were empty in 15 days. An infected antrum without mucopus may be responsible for the symptoms in the type of case which we all see from time to time, in which the patient complains of nasal symptoms and we are unable to find any physical signs on which to base our diagnosis. It must have been the experience of all of us to have relieved such a patient by puncture of an antrum which appeared normal.

The formation of mucopus depends to some extent on the infecting organism. The pneumococcus was only present in the antrum on one occasion without mucopus also being present. The staphylococcus, on the other hand, was never present in pus. Certain organisms are present sometimes with pus, sometimes without, and the conditions under which mucopus are formed are not clear.

Originally it was intended to repeat the antrum culture in all the cases, but nearly all the cases improved so much that it did not seem justifiable to submit them to further antrum puncture. This improvement was seen not only in the clinical condition of the patient but also in the radiographic appearance of the antra, although few of them had become quite normal. This improvement must be attributed largely to the introduction of lipiodol into the antrum. Antrum culture is the only means of proving infection in an antrum and should be employed more than it has been in the past as a method of diagnosis.

In the discussion that followed the presentation of this paper, A. W. Proetz suggested that the patient should be placed in the upright position and that the X-ray beams should be horizontal. He had also found that if the lipiodol was diluted up to half with olive oil, making it less viscous, the sinus would empty not in 14 days but in 96 hours, and this more rapid emptying might make the method more practicable.

Carlo Monti,[3] from Italy, discusses the development of the nose and sinuses in the newborn and in children up to 8 years of age (as reported by Ormerod).

The frontal sinus is not obvious up to 2 years, and at the age of 6 years is the size of a green pea. The maxillary antrum is present at birth, increases till 3 or 4 months of age, and then remains much the same until the fifth year.

The ethmoid cells are present at birth and grow but little until about the fifth year. The sphenoidal sinus is present at birth as a very small depression and grows steadily throughout childhood.

The author quotes Bouchet as saying that true sinusitis is rare before the sixth or seventh year, and says that the so-called sinusitis of infancy is in reality an osteitis, beginning around the tooth germs and spreading to the upper jaw as an osteomyelitis. From one to four teeth may be involved and there may be considerable sequestration. Treatment of this condition should be limited to a small incision to let out pus and to the extraction of the diseased tooth germs with a curette.

The evidence of sinusitis in children is overlooked nine times out of 10 when the child has only a little catarrh in the postnasal space. When complications occur, they are found in nine cases out of 10 in the orbit. Le Mee is quoted as saying that 80 per cent of children with the adenoid syndrome have some sinusitis and that the adenoiditis is secondary.

The author suggests that Proetz's method of displacement should be more widely used in children, both for diagnosis and for treatment.

[Largely owing to the pioneer work of Dean, the existence of sinusitis in children at early ages has long been recognized on this side of the Atlantic. The statements in the last paper show that in at least one European country the facts so forcibly presented in the first two papers have not yet received adequate recognition. Even in America there are pediatricians and laryngologists who are unwilling to recognize these facts. Many still refuse to acknowledge that sinusitis exists unless there be empyema. Such would do well to carefully read the facts presented by Bowen-Davies. Some sinusitis in infancy may be osteitis, but empyema also occurs. It is the absence of pain in young children which is partially responsible for the nonrecognition of sinusitis in the young.—ED.]

Crooks[4] points out that the medical profession in Great Britain has been slow to recognize the importance of sinusitis in children. In discussing the causation of such sinusitis, he notes that antrum infection from bad teeth rarely occurs in children, but that upper respiratory infection is the usual cause. With every nasal cold there is sinusitis. A radiograph taken during a cold will almost always show some thickening of the mucous membrane in the antra. [In my opinion, this is an overstatement. I have found the antra of many children with colds uninvolved by clinical examination, by transillumination, by X-ray, by aspiration and by culture, so that I believe severe rhinitis without sinusitis does occur.—ED.]

The symptoms of acute sinusitis are severe headaches, localized pain, tenderness and an increase in fever. He mentions the not rare great edema of the orbit secondary to ethmoiditis in young children, and emphasizes the importance of the recognition of the nature of the illness because drainage of the ethmoid results in recovery, while nonrecognition may result in cavernous sinus thrombosis [or osteomyelitis of the frontal bone.—ED.]. [But he does not comment on how this complication occurs very often without the young child complaining of either headache or pain. The younger the child the more true this is. Some cases of this recover with hot applications and sulfanilamide prescribed by the pediatrician. All such should, however, be under the active care of a rhinologist. It is only one who can see into and treat the nose of the small child who knows what progress the disease is making.—ED.]

In the great majority of instances, the ostium of the antrum can be made to open and let out the discharge without surgical intervention. Among other aids to treatment, he mentions favorably the benzedrine inhaler. [In my opinion, this apparatus and this drug should rarely be used, and when used be used with caution — and never used for children.—ED.]

The signs and symptoms of chronic sinusitis are nasal discharge, frequent colds, cough, snoring and mouth-breathing, sore throat, enlarged cervical glands, headaches, otitis media and general debility. He remarks on the mechanism by which chronic sinusitis may give rise to lower respiratory infection. [Headache is a frequent accompaniment of sinusitis in older children. The younger the child the rarer this symptom. So

true is this that the complaint of headache in a child of 7 years or less warrants serious consideration.—ED.]

Diagnosis, treatment and prognosis are clearly and adequately described. Treatment in the first place should be preventive. Antrostomy is reserved for chronic suppurative sinusitis. In the young, the opening cannot be made very large unless the inferior turbinate is sacrificed, and the hole in the young bone is apt to close in a month or two. [An antrostomy opening in a child should remain open only long enough to permit the antrum lining to become normal. After that, its closure is an advantage. The inferior turbinate need not be sacrificed or reduced. It can be displaced inwards and upwards during the operation and then pushed back into its usual position.—ED.]

It is possible to cure inflammation in a sinus completely by adequate treatment, but, of course, the sinus remains and so does the inherent liability to infection of it which caused the original trouble.

Regarding the relation of the tonsils and adenoids to sinusitis, he states that the broad conclusion is justifiable that disease of the tonsils and adenoids is not a common cause of sinusitis and that their removal is neither preventive nor a cure of the condition. He thinks that it is possible that inflammation in the sinuses may give rise to infection in the adenoid and the posterior halves of the tonsils. He comments that some children may have had their tonsils and adenoids removed for symptoms which should more properly have drawn attention to the sinuses. Even worse, they may have developed sinusitis as a result of imperfect technique during the operation of removal of tonsils and adenoids.

In the same connection, Leathart[5] shows how conservative measures adopted at the Royal Liverpool Children's Hospital in preference to the radical removal of adenoids yielded excellent results during the last few years. In 70 per cent of those treated thus, cases of enlarged tonsils and adenoids with nasal obstruction were transformed to cases of enlarged tonsils and adenoids only. The treatment is the administration of small doses of potassium iodide over a period of three months, 1 to 3 gr. once a day, according to the size of the child. Potassium iodide causes an increased secretion from the mucous mem-

brane of all the nasal sinuses, as is evidenced by the greater amount of watery discharge seen in cases of catarrhal sinusitis. Given over a period of at least three months it is an excellent "nasal aperient." [It would be necessary to keep patients undergoing this treatment away from the allergist! —ED.]

In their review of the literature on allergy, Duke and Kohn[6] state that: "Stoeffer and Cook report on the result of treatment of intractable asthma in children with hypertonic dextrose solution administered intravenously. They used 150 to 200 cc. of a 25 per cent solution with an excellent clinical result, which they believe was due to a specific action of dextrose rather than to the effect of a hypertonic solution."

In the opinion of Albert H. Rowe,[7] "nasal allergy" as a term will probably replace many of the former terms, such as "hay fever," "vasomotor rhinitis," "hyperesthetic rhinitis," "paroxysmal rhinorrhea," "nasal neurosis," "nasal hydrorrhea," "catarrhal rhinitis," "spasmodic coryza" and "perennial hay fever." At present, a comparatively small group of these sufferers remains whose symptoms resist allergic therapy. For a condition presenting such symptoms, the term "vasomotor rhinitis" may be appropriate, but no condition should be thus classified without thorough allergic diagnosis and treatment.

Nasal allergy in children is very common. It produces congestion, nasal blocking, sneezing, itching, mouth-breathing, clearing of the throat, hacking, snorting, noisy breathing, pushing, picking and rubbing the nose and restless sleep. [Note the difference from the symptomatology of sinusitis given above.—ED.] Bacterial allergy rarely occurs. The infrequency of complicating sinusitis in children is striking.

Differentiation of nasal allergy and nasal infection is most important. Nasal infection produces a red rather than a pale, boggy mucosa, polymorphonuclear white cells rather than eosinophiles in the nasal secretion.

The frequency with which in children attacks of nasal allergy and especially of asthma are initiated by sneezing coryza and allergic nasal congestion which suggest an infectious cold has led to the erroneous idea that infections cause such attacks.

Nasal allergy produces a pale, edematous mucosa of varying redness. This increases when active infection is present. Occlusion may be severe since the tissue is of a loose, erectile type. With continued swelling, the mucous membrane thickens and polypoid degeneration may result, owing to outpouching of the membrane, caused by closing of the glandular orifices. Such chronic allergy is one of the common causes of hyperplastic rhinitis even in the ethmoid sinuses.

Allergic edema is usually greatest on the lower edge of the middle turbinated bone [middle turbinal—ED.] in the middle meatus, at times on the anterior edge of the inferior turbinated bone and over the tubercle of the septum. [These conditions can only be seen by a rhinologist. Localized edema is due to near-by inflammatory swelling — not to allergy.—ED.]

It is important to realize that both infection and allergy may produce acute or chronic rhinitis, sinusitis, pharyngitis, tracheitis, bronchitis or laryngitis.

The absence of fever and malaise, the presence of a red rather than a pale, boggy nasal mucosa [surely, he means the presence of a pale, boggy nasal mucosa rather than a red mucosa—ED.], the history of the symptoms and, particularly, the presence of eosinophiles with comparatively few polymorphonuclear cells in the nasal mucus, favor a diagnosis of allergy.

Diagnosis rests on: *1.* History — sneezing, nasal blocking and watery discharge in the mornings. *2.* Other allergic disturbances in the patient or his progenitors. *3.* Pale edematous membrane, with or without polyp. *4.* Positive cutaneous reaction. *5.* X-rays of the sinuses may reveal thickened membrane, polypoid degeneration or varying opacity. The study of such mucosal swellings can be made with instillations of iodized poppy-seed oil. This seems necessary only in selected cases and is contraindicated if iodine allergy is demonstrated by the ingestion of potassium iodide for a day or two. The transient edema in the antral mucosa from the temporary effect of an allergin must be remembered. It is also recognized that these X-ray findings may arise from either infection or allergy and that they may indicate past difficulty which has been controlled.

In fact, the diagnosis of allergy usually cannot be certain until the results of therapy conducted for at least several weeks are determined. In other words, the final diagnosis usually must depend on the therapeutic tests. [It would be wiser to have an immediate examination by a rhinologist. The allergist is quite incompetent to examine the nose of a child. —ED.]

The relative number of eosinophiles or of polymorphonuclear cells in the nasal secretions indicates the relative amount of allergy or of infection present. If allergy is indicated, careful studies must be conducted in order to find the specific allergenic causes.

In cases of nasal allergy, surgical therapy is indicated when there is actual nasal obstruction which is not due to allergic edema. [Only the rhinologist is capable of deciding this.—ED.]

It is remembered that in most cases nasal blocking associated with so-called vasomotor rhinitis is due to allergy and if proper allergic therapy is instituted, the patient can usually be spared subjection to shrinking and intranasal treatment. [No rhinologist treats vasomotor rhinitis if he can get someone else to do it for him.—ED.]

Long-standing nasal allergy, however, as has been stated, produces structural changes irreversible in type which are greatly benefited by surgical treatment after allergic treatment is well established. [It would have been well to omit the words "after allergic treatment is well established." I have seen death of bone which occurred from pressure while allergic treatment was getting well established and while the patient was being kept away from the rhinologist.—ED.]

It is necessary to emphasize the conclusions of Dean, et al.,[8] if a successful result in chronic vasomotor rhinitis can be secured by treatment on an allergic dietary or endocrine basis, or by the correction of septal deformities, or by the removal of adenoids, or by the eradication of infection, the desired result will be secured without deleterious changes in the mucous membrane of the nose.

When one resorts to ionization or to the use of phenol, trichloracetic acid, alcohol or the cautery, one produces deleterious changes in the nasal mucosa.

H. V. Forster[9] points out that in these days when milk is being distributed freely to school children, many of them are sensitive to it, so that it might arouse vasomotor attacks in the nose.

This leads us to a few remarks on asthma. Creasy[10] reports on the rôle that the surgery of the paranasal sinuses plays in the asthmatic child. Sixty children between the ages of 5 and 14 years presenting the asthmatic syndrome in association with paranasal sinus infection were studied. Various forms of surgery were used and the surgery was adapted to the condition found. He is convinced that conservative surgical correction of structures within the nares and reventilation of the infected sinus cavities will undoubtedly assist in the rehabilitation of the health of the asthmatic child and his eventual cure. [Intranasal and sinus surgery on asthmatic patients cannot be too conservative — see what follows.—ED.]

Rackemann[11] reports, with permission, some findings of Dr. Mallory at the M.G.H. of the bronchial findings in a series of patients who died in an acute attack of uncomplicated asthma. The lungs were distended and thick and precisely like the lungs of guinea pigs dead in anaphylactic shock. The lungs form a perfect cast of the chest cavity. On cut section one sees the formation of tough, sticky plugs which protrude beyond the cut surface and which can be grasped with forceps and pulled out in a long, stringy mass. The clinical evidence that this plug formation is the true lesion in asthma is strong. Lipiodol shows the opaque material to extend readily to the medium sized bronchi and there to be sharply obstructed. The essential lesion appears to be a hypertrophy of the mucous glands in the bronchial wall.

Why do the bronchial mucous glands become hypertrophied and over-active? It is conceivable that a local allergic reaction taking place in the glands could in some way cause the stimulation. On the other hand, glands can be stimulated readily by nerve action, and one thinks of some nerve mechanism reflex in character. The close relation of asthma to disease in the nasal sinuses suggests this, but this relation is so close that I for one like to think of the sinus lesion which simulates the bronchial lesion so closely as a part of the picture rather than as a cause of it. I suggest that whatever the fundamental disturbance in asthma may turn out to be, it is something which

affects the bronchi and the sinuses at the same time. Operations on the sinuses do good in a few cases; that is true, but usually the effect is temporary only and *it is always possible that the good effect depends upon the nonspecific disturbance of the operation itself.* [Italics by Ed.].

The present status of the asthma problem is summarized by a recent remark of Louis Webb Hill: "The removal of those allergens to which a patient is found to be clinically sensitive is the surrender to a bad situation rather than a direct attack upon it."

According to Faulkner,[12] allergy is so protean in its aspects that it invades the realm of almost every specialist — thus, beyond all others, the rhinologist may be suspicious of all his cases which manifest any evidence of nasal obstruction. He will not usually lack sufficient warning from the allergist if he shows a propensity to operate on noses that are possibly allergic. In fact, if the rhinologist reads too many books on allergy he may develop a fear complex toward ever doing any nasal surgery.

Let us now consider some of the many pathological processes in the nasal cavities and the sinuses, where the allergist and the rhinologist must co-operate to insure proper diagnosis and treatment. The seasonal hay fever cases will present slight difficulty in diagnosis. If symptoms prevail in these cases beyond the hay fever season, one must always be suspicious of engrafted sinus infection. Most of them tend to get well, however, as the allergy subsides, and the nose acquires thereby better ventilation and drainage. A little judicious intranasal treatment will often facilitate this.

The sporadic cases with symptoms like acute nasal allergy often present considerable difficulty in diagnosis. Such cases present themselves at any season of the year, and while they may be a true allergy there are a great many which are not. A careful nasal examination may reveal a purely local cause for such attacks. If occurring after a coryza, one may be suspicious of retention of pus in one or more ethmoid cells. They should be treated by a thorough shrinking of the upper part of the nose. A suction irrigation of warm saline used afterwards will often dislodge several casts of ethmoid cells and the symptoms will clear up rapidly.

Other cases may be due to points of near contact in the nose between the middle turbinate and the bony spurs of deflections. These patients may have no symptoms until slight swelling of the membrane occurs, and then the tickling sensation begins. The swelling may be caused by entering a warm room, or even the slight nasal congestion associated with an overloaded intestine, mild indulgence in alcohol and numerous other factors. The areas of contact which produce these symptoms later on, with the membrane becoming more thickened, often give rise to constant headaches. A single application of cocaine will relieve most of these patients for some time, but a permanent cure is effected by either removing a portion of the middle turbinate or by a submucous resection of the septum, or both. I can recall many cases of this nature which had been previously diagnosed as allergic.

The conditions which give the most trouble in diagnosis are those where clinical evidence is not certain as to the presence of allergy and other obscure causes may be operating to produce symptoms.

If we now assume from picking up a definite clue from the history that we are on the proper trail of an allergic symptom complex in our own field, we should inquire about the time of day when symptoms are most aggravated. Nearly all allergic symptoms are worse at night and some produce this discomfort almost entirely in that period — examination of smears from the nose for the number of eosinophiles and their numerical percentage in the blood should also be determined. The nasal smear will likely show a far greater number of the eosinophiles in allergy than will be found in a smear from an infected process.

Let us now assume that we have a patient with a positive allergic history and that all tests were positive, we then proceed with our complete nasal examination. In mild cases, there will be found the usual signs of enlarged turbinates and general nasal obstruction. If the nose is wet and subject to frequent coryza attacks, with sneezing and free watery discharge, we would be well advised to refrain from all treatments except the usual palliative measures. The nature of the allergy in such cases must be determined and must guide the treatment. Even though the nose shows marked anatomical

defects, it had better be left alone until the allergy is brought under control. There is, however, a type of case much more chronic in nature with a large turbinate and very little discharge. These patients often complain of very frequent exacerbations when they think they are taking a fresh cold. The allergy in such cases should always be treated, but one can often do a great deal for them by minor surgical procedures, undertaken, of course, during a quiescent period. Such cases will often get such a satisfactory result that one may afterwards have doubt as to whether they were allergic after all.

The next type of case to consider is the more pronounced hyperplastic type with polyp formation, in which the history and tests are positive for allergy, although there are no active manifestations of allergic phenomena. Should the polyp not recede under requisite allergic treatment, it should be surgically removed as thoroughly as possible. These cases will need very careful after-care and any recurring polyps should be removed at once. In spite of this, unless the allergy is brought under control there will be some recurrence.

The question whether all polyp formations are associated with allergy is much debated. Faulkner finds many polypi which show no general evidence of being allergic. He goes on to say that such polypi do not tend to recur after removal to the same extent as the allergic cases. He can recall many cases where there has been no sign of recurrence over a period of years. Another reason for believing that such polypoid cases are not necessarily allergic is the fact that they have been totally relieved of symptoms and permanently cured by surgical methods. This, of course, does not prove that they are not sensitive to their bacterial proteins which disappeared with the eradication of the focus of infection, but many present only symptoms of a chronic infection.

It is very common in the routine of nose practice to find the two factors on allergic diathesis with a superimposed infectious process. Both factors should be treated — and it is just as well to give the allergist a chance to do what he can before any attempt at surgery is undertaken. If bacterial allergy is suspected, a long course of vaccine treatments may be worth trying. He thinks the only result he had ever seen with vaccine in sinusitis is one of desensitization. This does not cure the sinusitis but may afford relief from symptoms.

The association of asthma with any of the above type of cases has led to a great deal of discussion. The majority of allergists believe that the intranasal polyp formation is merely an association and not related as cause and effect. They are inclined to condemn the futile attempts to cure asthma by intranasal surgery. . . . There is also a great discrepancy in reported results by rhinologists. This can be partially explained by the variable quality of intranasal sinus surgery, but even in the most skillful hands there is a large percentage which fail to improve.

He is certain that he has never done radical surgery in a nose with polypoid degeneration associated with asthma in which he did not get some improvement, with an occasional cure. Since these cases are miserable from the local discomfort of a blocked-up nose, he thinks one is justified in clearing them out. Though one can never promise very much about curing the asthma, he doesn't know of anything else that offers even as much promise as surgery in obstinate cases.

The pathology of certain very severe and disabling headaches is a thickened membrane in the ethmoids and sphenoids, even completely polypoid with no sign of polypi in the nasal cavities and either no discharge or a small amount of postnasal mucoid drip. It would seem that polypi within the cells can exercise enough pressure to give pain, whereas polypi that have broken through the cell walls and filled up the nasal cavities are very seldom associated with any pain. The X-ray will show this condition very clearly, especially one taken in the verticomental position, and is better evidence than one can obtain from the most careful clinical examination. In fact, some cases examined after cocaine applications show practically nothing. Patients will complain of some nasal obstruction, worse during the pain exacerbations, and perhaps of some postnasal discharge. The allergy usually seems to be the dominant factor, though it is probable that infection was the initial cause. When the intracellular membrane has undergone such a change, antiallergic treatments will give no respite and surgery offers the only promise of relief.

He concludes a valuable paper by making a cynical observation about some of the ultraconservative opinions regarding sinus surgery. Honest statisticians are not always good sur-

geons and from a long experience with secondary sinus operations he feels that there are very many of them badly done. Incomplete operations may do more harm than good and merely stir up an infectious process without removing it.

Fletcher[13] says that sinus infection is so common that few people escape it. In the past, attention has been focused almost entirely upon the extreme case of sinus infection in which the sinus is filled with pus and drainage is completely obstructed — in other words, empyema of a sinus. In his experience, this type of case where surgery is the prospective treatment occurs only once in about 100 cases of sinus infection. His article attempts to explain how sinus infection may be diagnosed without any other aid than the ability to take the history and discover the presence of the existing symptoms.

Examination of the sinuses should be made during an attack of an acute infection and again when the patient has recovered. When recovery has taken place, with the sinus free from fluid and the mucous membrane recovered from the infection, transillumination or X-ray pictures may not reveal evidence of the past infection. It is important to keep this in mind when examining these patients; however, it is not uncommon to examine patients years after they have had sinuses and find by transillumination and by the X-rays the clouding of the sinuses. This may simply be the scars of the disease long ago cured and the patient may not need treatment.

In his opinion, to recognize "a cold in the head" is to make a positive diagnosis of acute sinus infection. He says that sinus disease is an all too commonly neglected focal infection, acting just as other focal infections, and that treatment for sinus infection is so simple that every family doctor can handle it well. [In my opinion, rhinitis and coryza occur often without any accompanying sinusitis, and sinusitis can rarely be diagnosed or treated by one unskilled in examination of the nose.—ED.]

Regarding "colds," the *Lancet*,[14] in an admirable editorial, says that well controlled tests of the efficacy of vaccine against the common cold have already shown that their value is at best highly questionable. Another nail is driven into the coffin of their reputation by an admirable study, lasting more than

three years, reported by Diehl, Baker and Cowan, of the Students' Health Service and the Department of Preventive Medicine and Public Health of the University of Minnesota. Their subjects were all students of the university, who volunteered to participate because they believed themselves to be particularly susceptible to colds.

They were divided into equal groups and three experiments were undertaken. They were all under the impression that they were having the vaccine, and even the physicians who saw them at the health centre when they contracted colds did not know from which group they came. Clearly, it would be hard to devise a better trial and the authors will be congratulated on its thoroughness.

This careful experiment supports the view that vaccines are of little or no value, either for reducing the incidence of colds or as safeguards against complications arising from them. They show the necessity of contemporaneous controls, for in the face of the results it is difficult to have any faith in histories of past attacks.

And, lastly, as the authors point out, they demonstrate how easily individual opinions can go astray: "During the course of this study several physicians have written or called us to say — I have a patient who was a student at the university last year and took your cold vaccine and got such splendid results that he wants to continue it. Would you be good enough to tell me what vaccine you are using? — and we would look it up and find in many instances that the person in question got the sterile solution or the lactose capsules. [Many are the treatments devised for the common cold, many of them most sincerely by physicians who recognize that their clinical impressions of the efficacy of remedies are often fallacious. Regarding these, there are two problems. Do any of these intranasal medicaments do any good; and, can they do any harm?—ED.]

In an attempt to answer these problems, Walsh and Cannon[15] present an illustrated and clearly written article which merits the close attention of all physicians — particularly pediatricians.

Popular advertising, as well as common practice, has made widespread the treatment of colds and nasal obstruction by

the use of nasal drops and sprays. Heretofore, physicians had assumed that even if such preparations did little or any good, they did at least no harm.

The authors discuss simply and with clarity: The effect on the nasal mucosa of materials used as vehicles for intranasal medicaments; the effect on the nasal mucosa of drugs used intranasally; the passage of nasal medicaments from the nose to the lungs; the effects upon the lungs from the intranasal administration of watery solutions of some commonly used medicaments; the effect upon the lungs of vasoconstrictive agents in watery solution.

Their article illustrates the early changes in the lungs of normal rabbits following intranasal instillation of oils and watery solutions of antiseptic astringents or nasal constrictors and they make the following general findings:

1. Oily solutions when instilled intranasally in small amounts in a healthy rabbit went quickly to the lungs and caused edema, desquamative alveolitis and focal lipoid pneumonia.

2. Watery solutions and antiseptics and astringents similarly entered the lungs and caused not only edema but severe focal necrosis, purulent bronchitis and bronchial pneumonia.

3. Isotonic saline solutions of such nasal constrictors as ephedrine and neosynephrin caused no significant degree of pulmonary damage after intranasal instillation in normal rabbits.

4. The potential dangers of intranasal medication are discussed. The conclusion is that the only completely safe medicaments are weak saline solutions of appropriate nasal constrictors.

The authors comment that objection may be made that even though they do so in lower animals, the materials used do not necessarily do so in human beings. This argument, however, fails with respect to the nasal oils, inasmuch as the increasing numbers of cases of lipoid pneumonia now appearing in the literature proves that such light fluids readily enter the lungs in both infants and adults. It would seem that too little attention has been given to the question of the rationale of intra-

nasal therapy with medicated solutions. Is there, for example, any substantial evidence that either the medicated oil or the aqueous solutions of antiseptic agents actually exert a significant bacteriostatic or bactericidal effect and, if they do, is there any evidence that they can get into or beneath the epithelium, where the infected organisms are growing? Is there any evidence, furthermore, that with the mucous blanket being renewed so frequently, the solution being instilled or sprayed once or twice a day will remain localized long enough to cause a therapeutic effect? Certainly, the experiments of Hilding cast much doubt upon this latter possibility.

The authors make it perfectly clear that their experiments do not deal with the mechanism whereby intranasal medicaments in the human being may reach the lungs. All they demonstrate is that when such materials get into the lungs of healthy rabbits they may be extremely caustic and harmful. The possibility remains that if such materials get into human beings they may cause similar harm; their use intranasally, therefore, should be attempted with a realization of their potential danger.

[An article demanding the serious consideration of every practicing otolaryngologist. The experimental animal is not the same as the human subject. The human patient can and will blow his nose. It is this ability which enables him to get upper respiratory relief from intranasal medication without lower respiratory damage. The infant and young child resembles the experimental animal in this peculiarity of ineffective nose-blowing. This article shows how intranasal medication in the infant and in the young child in bed is attended with danger.—ED.]

[Granted that medicaments in mineral oils may produce the damages mentioned, it is equally certain that the instillation of lipiodol (a medicament in a vegetable oil) into the bronchial tree has been followed in certain conditions not by pneumonia but by improvement. —ED.]

Ephedrine is used so widely by nose and throat specialists that this in itself is sufficient excuse to refer them to a reliable article describing this drug that they are using and its uses. Gaddum[16] points out that ephedrine has a number of peculiarities. One of these is that if the same dose of ephedrine is

given repeatedly, the effect diminishes with each successive dose. This immunity lasts for only a few hours. The toxic effect ascribed to overdoses of ephedrine are: general nervousness and insomnia and tremor, vomiting and sweating, palpitation, urinary retention and skin eruptions.

Zinc sulphate was widely advertised as a preventive of poliomyelitis. This was the subject of a well conducted large field experiment in preventive medicine. An editorial abstract by the *Canadian Medical Journal*[17] of the valuable paper so produced is quoted in full:

"An outbreak of poliomyelitis which occurred in Toronto during the months of August, September and October, with an incidence rate of 1.1 per 1,000 population, afforded an opportunity for a trial of a nasal spray containing 1 per cent zinc sulphate, 1 per cent pontocaine and 0.5 per cent sodium chloride. In the spraying, 0.5 to 1 cc. of solution was introduced into each naris, and the spraying was done on two occasions, with an interval of approximately 12 days. The spraying was done according to the technique of Peet, Echols and Richter, but differed from their recommended procedure, in that it was not administered on three successive days, since it was considered that it would not be practical to do so. The work was done by the attending otolaryngologists, 44 in number, of eight hospitals in the city. Ninety-seven clinics were held. The staff of each clinic consisted of an otolaryngologist, a graduate nurse as assistant, a clerical assistant for recording the spraying, a public health nurse for recording personal data concerning the child, and an undergraduate nurse for taking the child's temperature. In the period of one week from the authorization of the study, 5,233 children had received the first spraying, indicating the success of the presentation to the public and the efficiency of the organization. The second spraying extended from Sept. 13 to Sept. 16, and supplementary clinics were held on Sept. 20, a total of 89 clinics being held. The work was conducted without the occurrence of any complications, the immediate after-effects seldom giving discomfort for more than 24 hours.

"A representative control group of 6,300 children was obtained in the city proper. As suitable control groups could not be obtained in the 12 suburban municipalities, 621 children

resident in these municipalities who received the two sprayings in the clinics and 158 in private practice were not included in the analysis. Included in the study, however, were 749 children who were sprayed by the same group of otolaryngologists in their private practice and concerning whom satisfactory data were obtained, making a total of 4,713 children resident in Toronto.

"Among the 4,713 children who were sprayed, 11 cases of poliomyelitis occurred to Oct. 12, 30 days from the second spraying. One of these occurred six days after the first spraying and was not included in the analysis. In the control group of 6,300 children, 18 cases occurred during the period. The attack rate in the period seven days after the first spraying to 10 days after the second spraying was 1.7 per 1,000 in the sprayed group, and 2.1 in the control group; in the period seven days after the first spraying to 20 days after the second spraying, 2.1 in the sprayed group, and 2.4 in the control group; and in the period seven days after the first spraying to 30 days after the second spraying, 2.1 in the sprayed group, and 2.9 in the control group. The differences between the attacked rates in the sprayed group and the control group were not statistically significant. In the suburban group, which included 621 children sprayed in the clinics and 158 in private practice, no cases were found to have occurred. In the total of the city and suburban groups, the attack rates for the period seven days after the first spraying to 30 days after the second spraying were 2.9 in the control group, and 1.8 in the sprayed group. This difference also was found not to be statistically significant.

CONCLUSIONS.

"*1.* This study furnishes no evidence of the protective value of a nasal spray containing 1 per cent zinc sulphate, 1 per cent pontocaine and 0.5 per cent sodium chloride, when 0.5 to 1 cc. of the solution was sprayed into each naris on two occasions, with an interval of approximately 12 days, the spraying being performed by otolaryngologists with equipment suitable for spraying the olfactory area.

"*2.* As the spraying employing the method used in this study must be conducted by otolaryngologists or other physicians

specially trained in intranasal treatment, requires special facilities and cannot be done sufficiently quickly to meet the emergency of an outbreak, it cannot be considered a practical procedure." (Tisdall, Brown, Defries, Ross and Sellers, *Canadian Public Health Journal*, 1937.)

C. G. Smith,[18] in the Department of Anatomy, University of Toronto, carried out experiments on rats to determine the effect of the zinc sulphate solution used as a prophylactic agent in the poliomyelitis epidemic. This short article is clear and illustrated.

The evidence obtained was that the three types of cells in the olfactory epithelium (the olfactory, sustentacular and basal cells) may be destroyed by 1 per cent zinc sulphate, and that replacement of only the nonsensory cells occurs.

It is pointed out that histologically the human sensory epithelium is indistinguishable from that of the rat.

[This is an adequate explanation of the impairment or the complete destruction of the sense of smell which has occurred in some patients. To my knowledge, a senior member of an academic medical faculty has had no return of the sense of smell in two years.—ED.]

Goodale,[19] in an analysis of 75 cases of bronchiectasis from the viewpoint of sinus infection, shows that the greater number of the cases had their onset early in life. Twenty-four had the onset in the first decade and 20 in the second decade.

McFarlan[20] reports a careful study made to gain an idea of the incidence of pathogenic staphylococci in the nose of individuals who are free from staphylococcal infection.

For a long time it has been agreed that in a healthy nose, cultures from the walls of the nasal cavity proper yield relatively few organisms, while the vibrissae and crusts of dried secretion yield a plentiful and varied growth. From the point of view of a possible source of infection of the patient's fingers, it is the vestibule which might be important. In this regard, it has been suggested that a factor in the chronicity of some cases of furunculosis and osteomyelitis is the transference of pathogenic staphylococci by the patient's finger from the nose to the skin or wound. Apart from the possibil-

ity of reinfection of a superficial lesion, it is conceivable that pathogenic staphylococci in the nose may sometimes gain access to the blood stream and give rise to an acute osteomyelitis or a perinephric abscess.

In the course of this investigation, 33 children in hospitals, between the ages of 7 months and 16 years (and a much larger number of adults), were studied.

Pathogenic staphylococci were isolated from the healthy noses of 58 per cent of the children in hospitals. Similar figures for the incidence in individuals with no staphylococcal infection were obtained by Hallman in a larger series in 1937. It is suggested that the great majority of healthy individuals are carriers from time to time. The isolation of pathogenic staphylococci from the healthy nose of a patient with chronic staphylococcal infection is not in itself proof that reinfection from the nose is responsible for the chronicity of the infection so that the possibility of such reinfection cannot be denied.

The value of physical methods in the treatment of suppurative conditions of the nose and throat was the subject of a Symposium before the Section on Laryngology of the Royal Society of Medicine. Skiagrams, taken before and after treatment, from cases of children, age under 14 years, who were suffering from bronchitis or asthma associated with opacity of the maxillary antra, were shown by Bauwens[21] without comment. Hill and Taylor categorically ascribed all the action of high frequency currents to heat alone. Zamora[21] stated it to be dangerous to consider that short wave therapy will do good even in the absence of drainage. There should be no delay in the application of vasoconstrictor drugs in the region of the infundibulum. In the absence of drainage, short wave therapy may lead to increased toxemia. He pointed out that Hollender's histological studies of mucous membrane after ionization conclusively proves that fibrosis takes place and is accompanied by a permanent loss of cilia, so that supporters of the ciliary function of defense would look with disfavor on that effect.

Effler[22] describes an unusual case of an "orbital abscess" in a boy, age 11 years. A large postocular abscess was evacuated at the first operation, with partial removal of the inner orbital wall and partial removal of numerous ethmoid cells and polypi

on the affected side. The wound was left open. In spite of this, polypi recurred in abundance, refilling the cavity made at the first operation. A few weeks after the first operation, a second operation was performed, consisting of a complete ethmoidectomy, complete exenteration of recurrent polypi, currettage of frontal and sphenoid tissues and closure of the external wound. The right antrum was not disturbed. The author shows a photograph of a satisfactory postoperative result.

Lewy[23] reports in detail a case of gangrenous osteomyelitis of the left maxilla, ethmoid and sphenoid in a child, age 11 years, which ended fatally.

Following scarlet fever, there was a discharge from the left side of the nose, associated with a swelling in the left cheek. After about a month of unsuccessful local and general treatment, a nasoantral window operation was performed, and a peculiar musty odor of the breath developed with fever. Three months later, there was a swelling in the left cheek extending into the lower eyelid and obliterating the upper part of the nasolabial fold, with, in addition, an ulcerated area about 5 x 5 mm. in the left side of the hard palate, covered with a whitish-gray exudate and not involving the bone macroscopically; the left upper canine tooth was loose and the left nostril was filled with greenish mucopus. Biopsy made at the time of the nasoantral window operation was reported as showing granulation tissue only. Three months later, a smear of the material from the ulcer showed a predominance of fusiform bacilli and spirilla.

A Caldwell-Luc operation revealed moderately thickened and edematous antral mucosa without any free pus. Exhaustive histopathologic examinations and bacteriological investigations were made but nothing conclusive was found. A special effort was made to culture the anerobic necrophilic hemolytic streptococcus but without success.

The treatment is given in detail.

Six months after the onset, the entire facial antral wall. the nasoantral wall, the left alar partition of the nose and part of the septum had come away by sloughing. Two months later, this horrible hole into the face was relatively healed over

and just a small area in the nasolabial fold remained open. The child's general condition had steadily become worse, and in spite of the greatest possible care and many different types of therapy, severe bleeding, requiring packing, recurred several times, one attack terminating fatally.

An interesting side phenomenon was that this child while in bed grew to nearly six feet in length.

The author refers to the literature where a number of such cases had died, where various names have been given to the above process.

Northington[24] reports how an incorrect diagnosis of tonsillitis and acute catarrhal middle ear infection was made on a boy, age 11 years. It was not until the fourth otolaryngologist had examined the patient that the disease present was suspected. His report was: "Soft palate immobile on the left side, large irregular mass, resembling adenoid, filling left nasopharynx, left drum retracted and slightly injected in upper part, left hearing moderately impaired, no mastoid tenderness. In spite of mastoid cloudiness in the X-rays, don't operate. Remove tissue mass in nasopharynx and get microscopical examination." It was a lymphoepithelioma and responded satisfactorily to X-ray therapy. He remarks that in the boy the disease would have been discovered earlier if recognition had been given to the long acceptable observation that unilateral disease of the pharynx is not caused by the ordinary infections of the throat.

Nelson[25] reports a nasopharyngeal fibroma which was resistant to radium and inaccessible for electrolysis.

A boy, age 9 years, was seen because of increasing nasal obstruction, frequent colds, apparently chronic nasal discharge of a few years' duration, frequent mild nosebleeds, frequent bronchitis and prolonged fever after colds. The mucosa of both sides of the nose was congested and there was some mucopurulent secretion, especially around the middle meati. A large mass resembling ordinary nasal tissue filled the posterior half of the right nasal chamber and extended around the posterior septal edge and was visible through the left side. Its origin was not visible; with a probe, it seemed to be at the posterior tip of the middle concha. The mass could be

moved slightly. It was somewhat lobulated, moderately soft, though not flabby like the usual polyp, and of the red color of nasal mucosa. Roentgenograms showed no signs of a bony tumor or of resorption; there was marked polypoid thickening of the right antral mucosa, and slight thickening of the left, and slight cloudiness of the left frontal sinus. Diagnosis was obviously nasal tumor but the type was uncertain. The impression was that it certainly was not malignant, bony, cartilaginous or actually fibroid in consistency, yet the color and consistency were not those of the usual nasal polyp.

At the first operation, a nasal snare was engaged at the base of the polyp and avulsion was attempted. The wire of the snare broke. Deflection of the tumor with a mastoid gouge, intranasally guided by the finger in the nasopharynx, revealed a dense, fibrous, well encapsulated mass invading the sphenoid body through its bony face. A mass about 2.5 cm. in diameter shelled out rather easily, but a lobule remained firmly attached to the lateral wall below the region of the sphenopalatine foramen.

The histologic report showed the mass to be composed of new proliferating fibroblasts and old, stationary fibrous tissue. The central areas showed both perivascular hyaline and deep myxomatous degeneration. The vascularity was moderate, and venous sinuses in places were greatly dilated and engorged. There was no evidence of malignant degeneration. The tumor was encapsulated. Diagnosis of benign nasal fibroma undergoing myxomatous and hyaline degeneration was made.

About six months later, recurrence was certain. Radium pack and radium needles were used. Finally, the growth seemed stationary, but a severe spontaneous hemorrhage occurred, lasting one hour, and the hemoglobin was reduced to 40 per cent, compelling the authors to resort to a second operation.

Two years after the original operation, Sewell's transantral approach to the pterygopalatine fossa was taken. A window 2.5 cm. square was cut in the postantral wall. The exposed blood vessels were ligated and further dissection led to the capsule of the tumor, which was grasped with a heavy tenaculum through the nose and rapidly shelled out with a blunt dissector. Three years later, there was no recurrence.

Hagens[26] describes a case of congenital dermoid cyst and fistula of the dorsum of the nose. At first, the patient had a pin point sinus on the dorsum of the nose, from which blood, yellow material, hair and watery fluid were occasionally discharged. At 2 years of age a bump on the forehead was followed by a swelling and abscess, which was incised. The latter failed to heal completely, so that from time to time there was redness, swelling and, finally, discharge. When the lesion on the forehead was active, that on the nasal dorsum usually remained inactive, and later the reverse would occur. At 4 years of age, he was admitted to a surgical service because it was believed he had osteomyelitis of the frontal bone. An attempt to outline the passage with iodized poppy-seed oil was unsuccessful as the material could not be forced into the fistula.

It was decided to attempt the removal of the fistulous tract of the forehead. A definite tract was removed and its lower constricted part followed to the root of the nose, where its further extension was lost. Healing occurred. Three months later, a second operation was performed and an attempt was made to remove a cystic cavity found at the site of the fistula on the nasal dorsum. A large part of the cyst was beneath the level of the nasal bone. The two portions seemed to connect through a round opening between the lower part of the nasal bone; the outer portion of the tract was removed, and an extension upwards to the root of the nose was followed as far as possible. Some secretion and a large number of black hairs were encountered. The deep portion of the cyst did not communicate with either nasal passage, although the intervening soft tissue was not especially thick. On account of the danger of establishing an opening into the nose, this portion of the tract was not excised, but was thoroughly curretted and treated with acetone-trinitrophenol, phenol and alcohol. The incision was closed and after a day or two of drainage the wound healed satisfactorily. At present, the site of the former fistula appears normal; there is little scarring.

The wall of the cyst was examined microscopically and a tract lined by stratified squamous epithelium revealed. There were hair follicles and sweat glands in the dermis. The diagnosis was dermoid cyst of the nose. Pictures are given illustrating the condition; also a review of the literature.

BIBLIOGRAPHY.

1. Ebbs, J. H.: A Note on the Incidence of Sinusitis in Children. *Brit. Med. Jour.*, Feb., 1938.

2. Bowen-Davies, A.: The Paranasal Sinuses in Children. *Proc. Roy. Soc. Med.*, 31:12:1411, Oct., 1938.

3. Carlo-Monti, P.: Diseases of the Nasal Sinuses in Children; Abstract by F. C. Ormerod. *Jour. Laryngol. and Otol.*, 53:9:614, Sept., 1938.

4. Crooks, James: Nasal Sinusitis in Childhood. *Brit. Med. Jour.*, April 30, 1938.

5. Leathart, T. W.: The Biological Significance of the Tonsils and Adenoids and Other External Lymphoid Masses. *Brit. Med. Jour.*, p. 835, Oct. 22, 1938.

6. Duke, W. W., and Kohn, C. M.: Review of Literature on Allergy. *Arch. Oto-Laryngol.*, 28:3, Sept., 1938.

7. Rowe, Albert H.: Nasal Allergy. *Arch. Oto-Laryngol.*, 28:1, July, 1938.

8. Dean, L. W.; Linton, L. D.; Smith, H. M.; Dean, L. W., Jr., and Mahoney, Charles: The Treatment of Allergic Rhinitis. *Jour. A. M. A.*, 108:251-258, Jan. 23, 1937.

9. Forster, H. V.: *Proc. Roy. Soc. Med.*, 31:3:226, Jan., 1938.

10. Creasy, Raymond C.: The Role That the Surgery of the Paranasal Sinuses Plays in the Asthmatic Child. The Laryngoscope, 48:6:415, June, 1938.

11. Rackemann, F. M.: Discussion on Certain Aspects of the Asthma Problem. *Proc. Roy. Soc. Med.*, 31:5:529, March, 1938.

12. Faulkner, E. Ross: Problems in Diagnosis and Treatment of Hyperplastic Sinusitis and Allergy. *Ann. Otol., Rhinol. and Laryngol.*, 47:1:144, March, 1938.

13. Fletcher, William: The Sinuses as Point of Focal Infection and the Treatment of Sinusitis. The Laryngoscope, 48:1:17, Jan., 1938.

14. Editorial on Inoculation Against Colds. *Lancet*, p. 951, Oct. 22, 1938.

15. Walsh, T. E., and Cannon, Paul R.: The Problem of Intranasal Medication. *Ann. Otol., Rhinol. and Laryngol.*, 47:3:579, Sept., 1938.

16. Gaddum, J. H.: The Alkaloid Ephedrine. *Brit. Med. Jour.*, p. 713, April 2, 1938.

17. Editorial on the Value of the Zinc Sulphate Nasal Spray in the Prophylaxis of Poliomyelitis. *Canad. Med. Assn. Jour.*, 38:1:60, Jan., 1938.

18. Smith, C. G.: Changes in the Olfactory Mucosa and the Olfactory Nerves Following Intranasal Treatment with 1 Per Cent Zinc Sulphate. *Canad. Med. Assn. Jour.*, 39:2:138, Aug., 1938.

19. Goodale, Robert L.: An Analysis of 75 Cases of Bronchiectasis from the Viewpoint of Sinus Infection. *Ann. Otol., Rhinol. and Laryngol.*, 47:2:347, June, 1938.

20. McFarlan, A. M.: Incidence of Pathogenic Staphylococci in the Nose. *Brit. Med. Jour.*, p. 939, Nov. 5, 1938.

21. Zamora, A. M.; Forster, H. V., and Bauwens, P.: Discussion on the Value of Physical Methods in the Treatment of Suppurative Conditions of the Nose and Throat. *Proc. Roy. Soc. Med.*, 31:8:883-896, June, 1938.

22. Effler, Louis R.: Orbital Abscess. The Laryngoscope, 48:3:214, March, 1938.

23. Lewy, Alfred: A Case of Gangrenous Osteomyelitis of the Paranasal Sinuses. *Arch. Oto-Laryngol.*, 27:1:91, Jan., 1938.

24. Northington, Page: Some Cases of Otolaryngological Interest That Were Seen on the Neurological Service. The Laryngoscope, 48:1:5, Jan., 1938.

25. Nelson, Roy F.: Nasopharyngeal Fibroma. *Arch. Oto-Laryngol.*, 27:3:313, March, 1938.

26. Hagens, E. W.: Congenital Dermoid Cyst and Fistula of the Dorsum of the Nose. *Arch. Oto-Laryngol.*, 28:3:399, Sept., 1938.

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MODERN VIEWS REGARDING THE ANATOMY AND PHYSIOLOGY OF THE VESTIBULAR TRACTS.*†‡

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If anywhere the Greek sentence can be quoted: "Everything is in a flowing state, or in discussion," then such is the case with the vestibular apparatus. Even its *stimulation* is in question. Is it an angular acceleration, in the sense of Breuer; or a push, as modified by Mach; or a static wave (stehende Welle), after Alexander Spitzer? This question cannot be definitely decided.

Perhaps *comparative anatomy* clarifies this question somewhat. In *lower fish*, we have lateral line system, *i.e.*, open channels, distributed all over the body, in a direct open connection with the likewise open vestibular apparatus, and the waves of the surrounding water represent its stimulation; every change of these waves, *e.g.*, when approaching a rock, changes the perception, and is followed by a change of the direction in which the fish is swimming. This paleovestibular apparatus (in the sense of Kappers, Huber and Crosby), *i.e.*, the lateral line system and the vestibular apparatus, partly disappears in *higher developed* animals, and what remains is a vestibular apparatus with three semicircular canals instead of two, and an utriculus which adds to the persisting sacculus, the only vestibulum of the lower animals. In this connection we must emphasize that the vestibular apparatus in the lowest developed fish is open, and does not close until we reach the selachii.

We can, no doubt, assume that the waves which stimulate the vestibular apparatus in lower animals act similarly in higher animals and man and, as many scientists believe, convey the impression of the direction of the body when at rest or in action (Alexander Spitzer). These wave movements, of

*Read at the Seventy-second Annual Meeting of the American Otological Society, Inc., Rye, N. Y., May 22, 1939.

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‡This was aided in part through a grant from the Rockefeller Foundation.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 29, 1939.

course, can be produced by different irritants, by turning round, by temperature difference, by electricity and also by a blow or the vibration of a tuning fork.

Which parts are *irritated or stimulated?* The three cristae ampullares, the macula utriculi and that of the sacculus. It is interesting that the macula utriculi shows three parts: one, specific for the utriculus; the second resembles the crista ampullaris; and the third resembles the macula sacculi. We, therefore, can assume that besides the semicircular canals (which, according to Favill, only influence two eye muscles), the utriculus also influences the eye muscles, as do the canals, That is proof that the utriculus has a variety of functions,

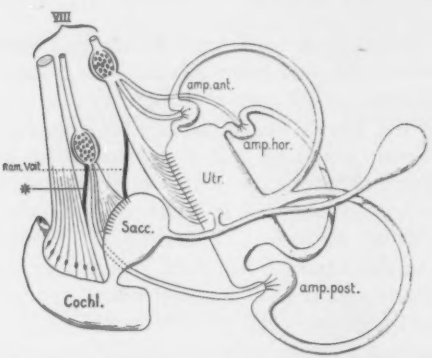

Fig. 1. Diagram showing branches of VIIIth nerve (Oort).

and the same variety is to be found also in the nerves and in the pathways (see Fig. 1).

Two nerves conduct these stimuli to the brain:

1. The *Nervus sacculoampullaris* lies quite close to the cochlear nerve; it was called ramus inferior by Retzius, or ramus posterior by Alexander, and it also includes the ganglion labyrinthicum (vestibulare) inferius. This nerve originates from the sacculus and the posterior semicircular canal.

2. The *Nervus utriculoampullaris,* or ramus superior of Retzius, ramus anterior of Alexander, originates from the utriculus, and from the ampulla anterior and horizontalis

(lateralis). It includes the ganglion labyrinthicum (vestibulare) superius.

It is perhaps better to designate, according to Alexander, the nervus sacculoampullaris and the nervus utriculoampullaris as the *Nervus labyrinthicus* instead of calling it nervus vestibularis.

Can we follow these two distinct labyrinth nerves into the *medulla* and into the *vestibular nuclei?* Former authors

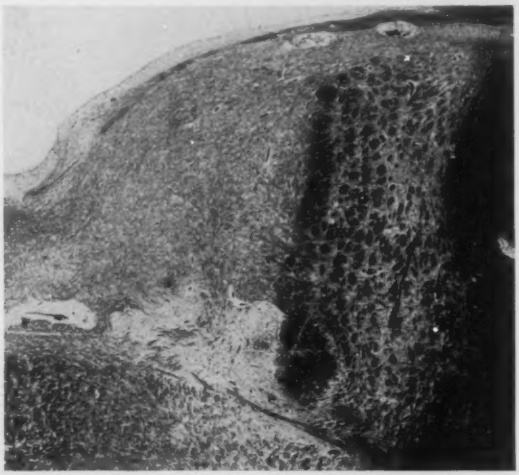

Fig. 2. Area fasciculata inside the restiform body (right). Between the bundles lies the nucleus labyrinthicus intrafascicularis, above the nucleus labyrinthicus suprafascicularis and medial from the bundles below the floor of the ventricle the nucleus labyrinthicus triangularis.

assumed three such nuclei: nucleus ventrocaudalis, triangularis and angularis.

1. One of them lies caudad medial from the restiform body (see Fig. 2) in the area fasciculata, and was called by Kohnstamm nucleus ventrocaudalis; I have named it (I believe more precisely) *nucleus labyrinthicus pars intrafascicularis.*

2. Cephalad, where this area is smaller, we find the same nucleus not only in this area but also dorsad from it (see Fig. 3). This dorsal part is commonly called nucleus triangu-

laris, although it is in reality only embedded in the most lateral part of the nucleus triangularis proper; therefore, I have called it *nucleus labyrinthicus pars suprafascicularis.*

I repeat: These two parts, pars intrafascicularis and pars suprafascicularis, form only one nucleus, which begins caudad near the hypoglossal region and ends cephalad near the abducens region.

3. The third nucleus of the former nomenclature with its position more cephalad was called nucleus angularis or Bechterew's nucleus. Since this nucleus also belongs to the nucleus labyrinthicus, we had better name it *nucleus labyrinthicus pars angularis.*

These three nuclei just described, namely, the nucleus labyrinthicus pars intrafascicularis, nucleus labyrinthicus pars

Fig. 3. Diagram showing the nuclei labyrinthici.

suprafascicularis and nucleus labyrinthicus pars angularis, form the essential *nucleus labyrinthicus.*

This nucleus labyrinthicus is accompanied (see Fig. 3) by the small, cellular *nucleus triangularis proper* formerly mentioned.

Besides these endings of the nervus labyrinthicus in the nuclei, there are also *direct vestibular fibres* which end in the cerebellum in its oldest part; namely, the *flocculus* and the *lingula* and *nodulus.*

It is a question whether there are other direct connections from the labyrinth nerve. We know that *collaterals* of the nerve enter into the *nucleus Deiters' magnocellularis.* It is possible that such fibres or collaterals also reach the reticular nuclei in the pons or medulla, and Leo Alexander and

Withacker found a connection with the red nucleus; it is questionable, however, whether these are direct connections.

Considering that the nervus sacculoampullaris is the more posterior one, near the cochlear nerve, we can conclude that it *enters* into the nucleus labyrinthicus pars *intrafascicularis*, and perhaps also into the posterior part of the nucleus labyrinthicus pars *suprafascicularis* and into the *nucleus triangularis proper*. Besides, it is certain that the fibres from the sacculoampullaris are the fibres laterad from the restiform body entering into *the flocculus* and into the nucleus labyrinthicus pars suprafascicularis (see Fig. 4).

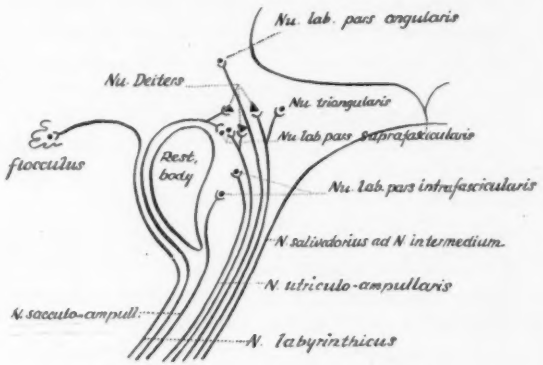

Fig. 4. Diagram of the nervus labyrinthicus.

The *nervus utriculoampullaris enters* into the *anterior* part of the *same two nuclei*, and into the nucleus labyrinthicus pars *angularis*, and also into the nucleus triangularis proper.

We must, however, bear in mind that every part of the labyrinth is connected with *more than one nucleus*, as Ewald stated. For instance, when moving the eye upwards we innervate not only the rectus superior but also the obliquus inferior. Favill explained this by the fact that, because of the joint entrance into the utriculus, stimulation of one canal also stimulates its fellow canal. Because of the fact that the rectus superior is also an adductor, we can understand that it can be influenced by all three canals. There is much evidence that

in the pathways from the labyrinth we have a close connection with several nuclei.

The *nuclear terminations* discussed are the main terminations, but it is most likely that there are also other connections with other nuclei.

For many years, beginning with the fundamental work in 1901 of Alexander Spitzer concerning the posterior longitudinal bundle, the *secondary pathways* from these nuclei have been under discussion. To mention only Muskens, Van der Schueren, Leidler and, in this country, Ferraro and Barera and my own investigations, it is certain that more fibres originating from the *nervus labyrinthicus,* except the angular part, are crossed, or at least *partially crossed,* and the amount of the crossed fibres is greater than the uncrossed. Only the fibres *from the angular part* remain, seemingly, on the same side. But when we examine more accurately we see that the fibres out of the nucleus labyrinthicus *intrafascicularis* and *suprafascicularis* cross caudally by way of internal arcuate fibres, while the others *from the angular part* cross partly cephalad by way of the posterior commissure.

The fibres *from the flocculus,* the Klimoff-Wallenberg fibres, run partly in the bracchium-conjunctivum and with it into the region of the eye muscle nuclei, where they end; partly, they run medial in the angle of the IVth ventricle into the region of the angular nucleus and the nucleus labyrinthicus suprafascicularis.

I must refer to a bundle originating from nucleus reticularis ventralis tegmenti pontis which is under the influence of the labyrinth: the *fasciculus pontis ascendens* of Lewandowsky. These fibres end in the oculomotor nuclei.

It is certain that these fibres from the flocculus, as well as those from the reticulated nucleus, are partly crossed. All these fibres just mentioned send branches *into the eye muscle nuclei;* namely:

1. Into the group for the horizontal movements, the *rectus externus (abducens)* and the *rectus internus,* both nuclei on the same side, whereas the fibres of the rectus internus are crossed to enter into the contralateral musculus rectus internus.

2. The second group is the rotatory one, chiefly represented by the *trochlear nucleus,* but also by the anterior group.

3. This third or anterior group, represented by the *rectus superior* and *inferior,* performs the movements upwards and downwards.

These two latter groups serve not only their own special movements but also the movements of all the other groups, while the first group only serves its specific movement.

This survey shows that *every eye muscle nucleus* can be influenced by the nucleus labyrinthicus intrafascicularis and angularis, while the *flocculus* and the nucleus *reticularis* pontis influence only the anterior group of the eye muscle nuclei. Therefore, I assume that the former connections innervate all the eye muscles. In any case, the connection of the nucleus labyrinthicus intra- and suprafascicularis is crossed, *fasciculus vestibulomesencephalicus-cruciatus.* And only a few fibres remain homolateral, *fasciculus vestibulo-tegmentalis* (Muskens).

But there is also a crossed part of this fasciculus; namely, the *fasciculus vestibulotegmentalis cruciatus,* better called thus than "medialis," as it was named by Muskens.

The connection from the angular nucleus remains, with reference to the eye muscles, homolateral: *fasciculus vestibulomesencephalicus homolateralis.*

When we add that the *tegmental fasciculi* also end in the nucleus interstitialis, the mesencephalic ones in the nucleus commissuralis, then we have surveyed all the *ascending fibres* for the *eye muscle nuclei.*

THE FUNCTION.

The oldest function of the labyrinth it seems to me is the so-called *skew deviation,* the Hertwig-Magendie *squint position.* As Jacques Loeb showed, it is still to be found in fish. When we change the position of a fish, turning it on its side, we get the skew deviation: the upper eye stands above and outward, the lower eye below and inward. Thanks to the courtesy of Dr. Breder, Director of the New York Aquarium, I could repeat these investigations and extend them to a lower

series of fish. For we know that the lowest developed fish, the cyclostomes, and some of the plagiostomes, have only one sacculus and one or two semicircular canals. Since I found that in these lowest developed fish there exists the skew deviation, we have the right to assume that this reflex is a *righting reflex* (see Fig. 5). Every change of position automatically also changes the position of the eye; the eye is fastened to the space so that the fish feels every change of its position.

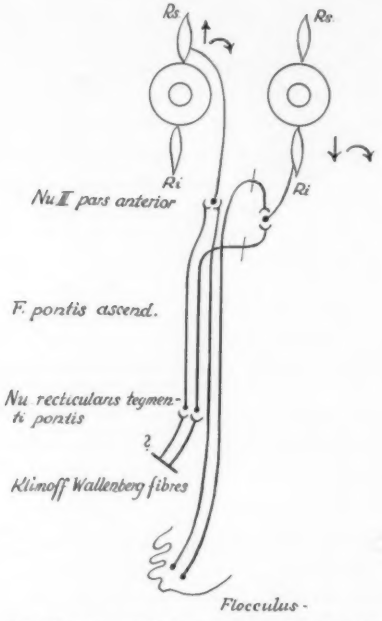

Fig. 5. Diagram showing the pathways and nuclei for the skew deviation.

Also, investigation of the flocculus by dissection, which was frequently performed, shows a skew deviation in rabbits.

In this case it is necessary to draw horizontal and vertical lines in the eye of the rabbit, as was done by R. Loewy in my Vienna Institute, in order to recognize this deviation. It is certain that the *upward movement* is caused by *homolateral fibres;* the *downward movement* by *crossed fibres of the flocculus.*

It is quite interesting that Mussen noted in animals with dissected flocculus a general disturbance of the righting reflex, which, however, was not corroborated by other authors.

This is one of the oldest functions of the labyrinth, and only this can be called a *paleolabyrinthic* reflex (Kappers, Huber and Crosby).

This skew deviation may also be seen *in man*, mostly in *lesions* of the cerebellar pedunculi, and, according to Poetzl

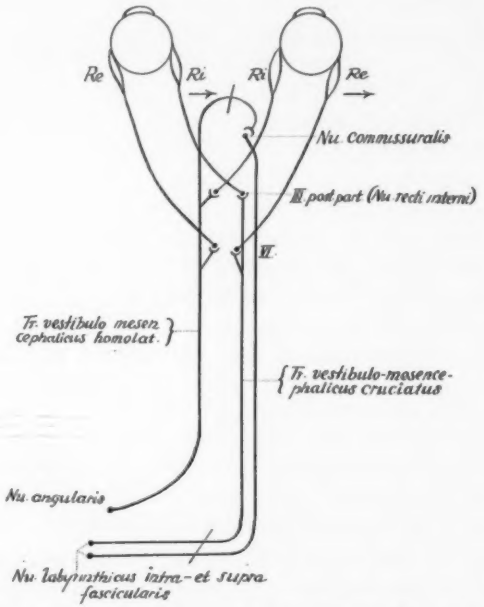

Fig. 6. Diagram showing the pathways for the associated eye movements to the side.

and Sittig, also in lesions near the posterior commissure. This is easily understood when we consider that the fibres of the flocculus end in the anterior part of the oculomotor nucleus.

The *neolabyrinthic reflexes* from the eye reach the nuclei by way of the above-mentioned pathways. For a better understanding, we must start from the *horizontal nystagmus* caused

by a lesion in the posterior part of the nucleus labyrinthicus (see Fig. 6).

There is a *quick* and a *slow component* of every nystagmus, a clonic and a tonic component, or a kinetic and a tonic component, respectively.

We know that the *clonic or quick component* of the nystagmus indicates the *side of the lesion* in the medulla and the pons. That depends on the fact that these quick movements are *innervated by the healthy side* of the labyrinth nuclei with their crossed fibres.

The *slow component* is *directed to the healthy side* because the tonic innervation is not crossed, since the fibres from the angular nucleus cross only in the posterior commissure, so that the tonic innervation of the healthy side has the preponderance.

I agree with Lorente de Nó that *the centres* for the *slow and the quick component* of nystagmus are not in the same place; but, as I was the first to describe, both components are to be found *in the same region* in the medulla; namely, the slow component in the angular nucleus and perhaps also in the reticulated nucleus; the latter was suggested by Lorente de Nó, and then thought probable by Spiegel, who cut both posterior longitudinal bundles and found nystagmus. We may conclude: The quick component of nystagmus depends on a lesion of the nucleus labyrinthicus pars fascicularis and its pathways; the slow or tonic component, as just mentioned, on a lesion of the angular nucleus or the nucleus recticularis ventralis tegmenti pontis and their continuations.

A second question is the *localization of the direction* of the nystagmus. I was the first who held that the rotatory and the horizontal nystagmus are caused by a more caudad lesion in the medulla, while the vertical nystagmus is caused by lesions near the colliculus. Leidler, Grahe and many other authors confirmed this statement. It is evident that, with regard to the position and the endings of the nerves of the semicircular canals, *the rotatory and the horizontal movements* are represented by the *nuclei behind the VIth nerves*, the nuclei *intra-* and *suprafascicularis* and *angularis*.

The discussion remains as to the nuclei for the *upward and downward movements*. A mistake is commonly made in dis-

regarding the righting reflexes to be found in the lowest animal series. With regard to this reflex, we must assume that the *flocculus* and the adjoining system of the fascicular nuclei produce the righting reflexes. *A bilateral lesion* of the fibres from the flocculus *cancels the shift to the side* so that we must get an upward or downward *paralysis* in these cases.

It is clear, however, that there must be another main centre for these two movements. My own opinion was that the labyrinth nuclei also give rise to fibres which run in the posterior longitudinal bundle and end in the anterior part of the oculomotor nuclei. And I thought that the *left bundle* conveys the fibres for the *upward movement*, the right for the downward, assuming it to be the same as with the lateral movements.

Muskens, on the contrary, suggests that the innervation of these movements comes from aqueduct nuclei; namely, from the mesial nucleus for the upward movement, and from the lateral for that downward. He thinks that the central tegmental fasciculus connects these nuclei with the inferior olive which receives vestibular fibres. But this conception is quite impossible for anatomical reasons.

Since Lorente de Nó drew our attention to the substantia reticularis as centres for the vestibular apparatus, many scientists endeavored to clarify these connections. It is a well known fact, mentioned in my atlas, that the posterior longitudinal bundle gets affluent impulses from the reticulated substances of the tegmentum pontis. One of these pathways is better known, arising from the ventral reticulated nucleus, described by Lewandowsky as *fasciculus pontis ascendens*, and ending in the anterior part of the oculomotor nucleus.

Godlowski could confirm this in a very interesting and explanatory work. He thinks that this nucleus and tract convey vestibular influences for the upward movements of the eye, while the downward movements are performed by the nucleus commissuralis (nucleus Darkschewitsch), the end of the posterior longitudinal bundle for its utriculoampullar part, so that this statement alone proves that the *posterior longitudinal bundle* is concerned *with vertical movements*. But Godlowski forgot the flocculus with its righting reflex.

Spiegel, Oppenheimer and Price, and Spiegel and Price could show by electroencephalography the significance of *cen-*

tres in the collicular region for the vertical eye movements without, however, deciding this question.

With regard to the certain connection of the anterior semicircular canal to the labyrinth nuclei, I maintain my opinion that the *labyrinth nuclei* and the *posterior longitudinal bundle* also innervate the anterior oculomotor nuclei, perhaps supported by the nuclei for the righting reflexes.

Easier to understand is the *optokinetic nystagmus.* One centre in the cortex (calcarina, parieto-occipital region, angu-

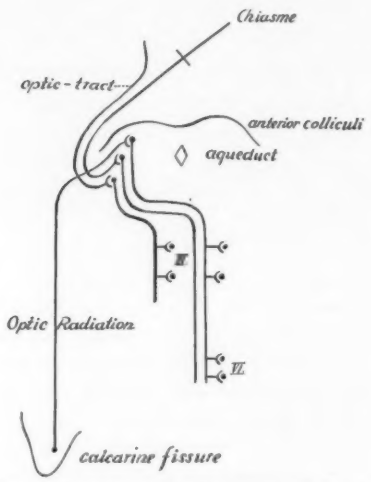

Fig. 7. Diagram showing the pathways and centres for the optokinetic reflexes.

lar region) (see Fig. 7) sends its fibres into the *anterior colliculus,* where Berl found them in the *stratum zonale,* while the other component, the optic nerve fibres, ends in the *stratum opticum* in the second layer, and here there is a magnocellular nucleus which gives origin to fibres turning in an arch, *the aqueduct,* to enter into the oculomotor nuclei.

Equally easy to understand is the *cortical innervation* of the associated movement of the eyes. I showed this many years ago, and I found that only the *pyramidal tract* can conduct such fibres in man, running to the pes pedunculi near

the pons, and from here the fibres enter *cephalad* into the *oculomotor nuclei, caudad* into the *VIth pair*, using, for a short distance, the posterior longitudinal bundle. Godlowski believes that this tract continues via the optic thalamus into the tectum colliculi. No doubt such tracts exist, but not for voluntary movements in man. We know that *fibres from the vestibular apparatus* also enter into the *optic thalamus*, partly interrupted by the commissural or interstitial nucleus, and end in the so-called *zona incerta*, via field H1 and H2, but also the ventral nucleus and the centre median.

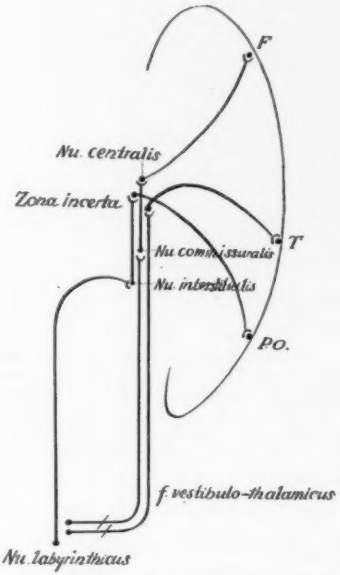

Fig. 8. Diagram showing corticovestibular connections.

This tract has *two functions:* the one, to bring *vestibular perceptions to the cortex* (see Fig. 8), in order to produce the *optokinetic* and the *voluntary movements*, the former by more ventrolateral fibres, the latter by fibres from the "centre median," for it is a mistake of Legros Clark to think that the "centre median" has no connection with the cortex. Sager and Godlowski were the first to show such connections in monkeys.

The second function of this tract is the *conscious perception* of this sense organ. Many authors, *e.g.*, Wallenberg and Held, suggest that such a pathway ends in the temporal lobe, as I showed in 1924, and Spiegel and his collaborator, Sala, proved later by electroencephalographic studies.

As to the *symptoms* produced by a *lesion of this bundle*, we cannot assume that *dizziness* is one of them, in spite of the fact that Spiegel and Alexander believe dizziness in tumors is a local symptom, found chiefly in temporal lobe affections, and almost as often in those of the *striopallidum*. I think

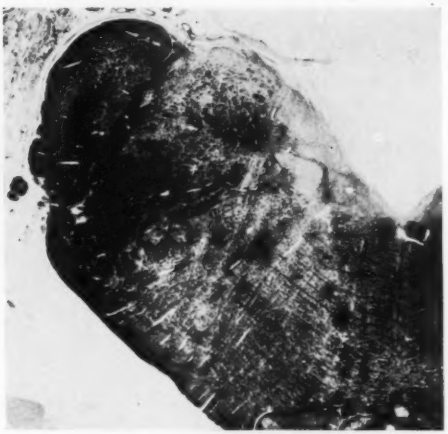

Fig. 9. Giraffe (from left to right): Restiform body, area fasciculata with the nuc. lab. intra- and suprafascicularis, nuc. triangularis. The pale focus embedded in this nucleus is attached to the IXth and Xth nucleus. It is surrounded by a bundle of arcuate fibres (dorsally).

dizziness is a more complicated symptom, caused by different disturbances in the vestibular apparatus. I adopt the opinion of A. Spitzer that the *conscious perception* of vestibular impressions means only the *impression of direction*.

Many years ago, in the Vienna Neurological Institute, studies were begun on the influence of the vestibular apparatus on the *vegetative system*.

Spiegel and his collaborators (Demetriades) and Alexander Spitzer assumed that the *triangular nucleus*, the proper tri-

angularis with the small cells, is a vegetative nucleus. Yet Winkler and Held described connections from this nucleus to the autonomic system.

I instituted comparative anatomical studies. My pupils, Takagi, Godlowski and Nakamura, busied themselves with these connections, and finally I, myself, summed up all the facts and found a direct connection *from the proper triangular nucleus* by way of the *fasciculus triangulointercalatus of Fuse*, which enters into the *fasciculus periependymalis* and

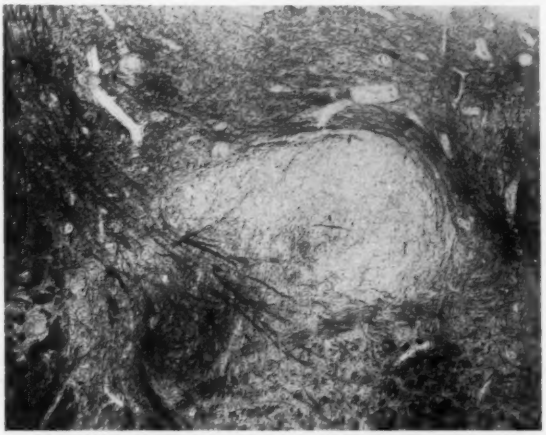

Fig. 10. Giraffe. Detail of Fig. 9 showing the distribution of the arcuate fibres with its three parts: dorsal horizontal fibres running to the periependymal tract and Roller's nucleus; then ventral fibres entering the vagus; glossopharyngeus nucleus and the reticulated substance.

from here into *Roller's nucleus*, ventrad from the hypoglossal nucleus.

To study the labyrinth neck reflexes, I chose the medulla of the giraffe, because this animal, due to its long neck, obviously must have a better developed labyrinth connection to the neck muscle centres (see Fig. 10). Here, this bundle is so well developed that I could distinguish *three parts:* One part in the vagus nucleus, the other part in the substantia reticulata, and the third part in Roller's nucleus. Even if all fibres do not have a vegetative function, most of them seem to be vegetative. A doubt is possible only as to the reticulated nucleus,

where fibres of the labyrinth for the labyrinth neck reflexes or labyrinth body reflexes may originate.

It is evident that Magnus and De Klejn find the centres for the labyrinth neck reflexes in the caudad half, because all fibres which *run in the spinal cord* must pass the *posterior part of the medulla* (see Fig. 11).

These fibres comprise: *1.* The *descending fibres* from the *nuclei labyrinthici. 2.* The fibres from the nucleus *interstitialis* and *commissuralis;* namely, the fasciculus interstitio- and commissurospinalis. *3.* Fibres *from the nucleus ruber,* the

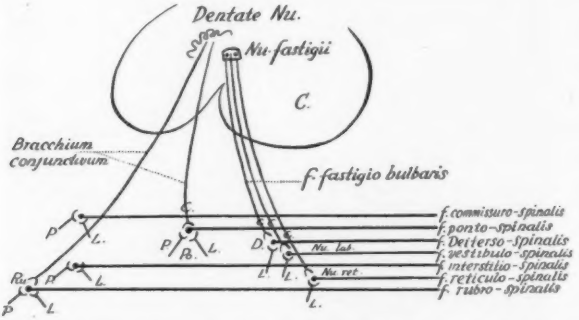

Fig. 11. Pathways from the vestibular-influenced nuclei to the spinal cord. (C) Cerebellum. (Co) Nucleus commissuralis. (I) Nucleus interstitialis. (D) Nucleus Deiters. (L) Fibres from labyrinth nuclei. (NuL) Nucleus labyrinthicus. (P) Fibres from pallidum. (Po) Pons tegmental nuclei. (Ru) Nucleus ruber.

nucleus *reticularis* pontis and medullae, and the nucleus *magnocellularis Deiters;* they are the rubro-, the ponto-, medullo- and the Deiters'o-spinal tracts. *All these latter fibres* originate from large motor nuclei, *influenced* on the one hand by the *vestibular apparatus,* and on the other by the *cerebellum* and the *stem ganglion,* a supplementary system for a part of the labyrinth reflexes, which we can call *paravestibular system,* rather than the term which Godlowski used.

But I suggest avoiding names as supravestibular system for all the nuclei and tracts beyond the nucleus labyrinthicus (according to Muskens) ; whereas Godlowski divides this system into two parts, one from the nucleus labyrinthicus to the

colliculi — the paravestibular system — and one part before the colliculi — the supravestibular system.

One of the most interesting questions has always been *the influence on the cerebellum of the labyrinth* (see Fig. 12). I cannot find that it represents a release apparatus. As in other apparatus, it influences the *degree of a movement* as well as the *degree of the tonus and the power*. We must not forget that one part of the labyrinth nuclei is in such intimate connection with the *cerebellum* that every *lesion of the latter* also injures the *labyrinth nuclei*, possibly only by way of the blood

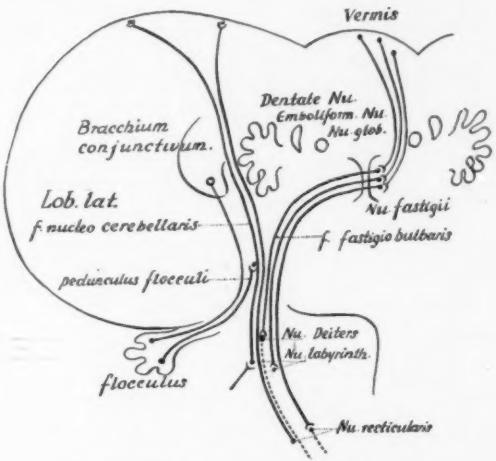

Fig. 12. The labyrinth cerebellar system.

vessels, because the posterior cerebellar artery supplies not only the lateral part of the medulla but also the cerebellum.

We have only one connection from the nuclei to the cerebellar cortex and back, via *nucleus fastigii* to the nucleus labyrinthicus; the first, the *nucleocerebellar* fibres; the second, the *fasciculus fastigiobulbaris*, after my nomenclature. In the same manner, the nuclei are influenced by the *stem ganglion* through the colliculi, and the so-called predorsal fasciculus.

We see that we have here the same connection, perhaps not so well developed, as in the nucleus ruber, nucleus resticularis

pontis and nucleus Deiters. And we can assume that these latter nuclei *supplement* the *righting* and the *tonic reflexes* of neck and body.

I do not believe that the cerebellum conveys impulses by way of the red nucleus into higher centres, as is supposed by many authors.

I mentioned already the pathways and centres for *righting* and *postural reflexes*. We have for the *righting reflex* (see Fig. 13) the *nucleus interstitialis*, connected by the fasciculus

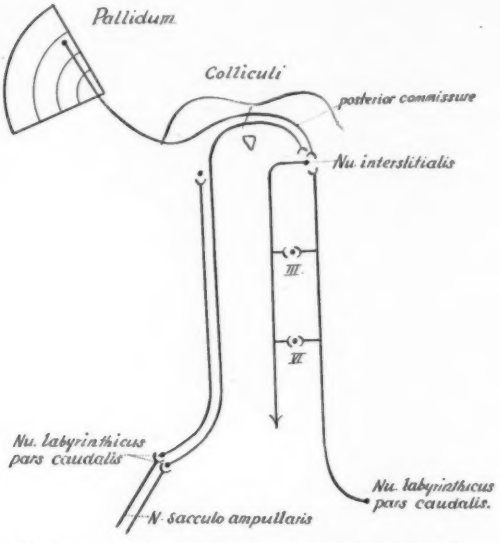

Fig. 13. Diagram showing pathways and nuclei for righting reflexes.

vestibulotegmentalis, crossed and uncrossed, with the vestibular nuclei, by fibres in the posterior commissure with lenticular nucleus. And from this nucleus interstitialis there runs, medial in the posterior longitudinal bundle, the tractus interstitiospinalis.

Besides this apparatus for the righting reflexes, *the nucleus commissuralis* receives fibres, crossed and uncrossed, from the tractus vestibulomesencephalicus, arising from the nuclei

labyrinthici fasciculares and angularis, and also connected by the posterior commissure with the lenticular nucleus (see Fig. 14). From this commissural nucleus the *tractus commissurospinalis* conveys *tonic impulses* caudad to distribute the tonus in the muscles for our posture.

CONCLUSIONS.

The first sense organ to be developed in animals, including man, was the vestibular apparatus, first joined with the lateral

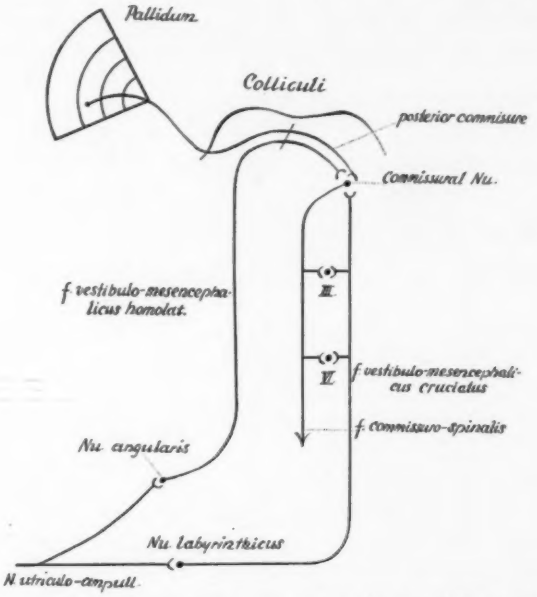

Fig. 14. Diagram showing pathways and nuclei for postural reflexes.

line system. It protects fish and other marine animals by its sensitivity to the most subtle disturbances of the surroundings: *paleovestibular system.*

When the *eye* gains the first place among the organs of sense, the vestibular apparatus is connected with all muscles which *move the eye*, in order to recognize (to use this much abused term) our living space. In the lowest developed fish,

every change of position changes the position of the eye at the same time in the sense of a *skew deviation*, a paleovestibular reflex, a genuine righting reflex, that means the eye is fastened to the surrounding space in every position.

Then we see in higher developed animals a finely graded apparatus, controlling not only the eye muscle movements in all directions but also the righting and the posture of the eye muscles and all those muscles which support their action, and finally all the muscles of our body. Despite the fact that all these movements and regulations are reflexes, therefore unconscious, we get in the highest animals and in man a connection of the vestibular apparatus with the *cortex cerebri*. I mean a direct connection as Wallenberg and Held pointed

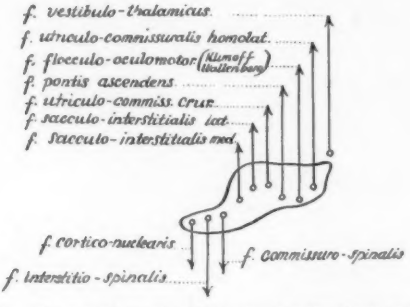

Fig. 15. The tracts in the posterior longitudinal bundle.

out, and not an indirect one via cerebellum and nucleus ruber. We get knowledge of all these occurrences; we obtain what we call *orientation* in space. Its disturbance, the disorientation, is called *dizziness*. The construction of our surrounding world is effected by the eye and its best support, the vestibular apparatus. In the same manner, we get knowledge of the movements of our body, first through a perception of *a change of direction*.

But this apparatus also influences the *vegetative system*, insofar as it is in connection with it; chiefly through the vagus system it influences the heart, the stomach and its appendicular organs to protect our life by regulating the function of these organs, as does the vestibular apparatus.

The labyrinth is not the apparatus for constructing our outer and inner world in the sense of Spitzer, but it supports this formation or, better still, it regulates this structure, to form a clear-cut picture.

BIBLIOGRAPHY.

ALEXANDER, GUSTAV: Makroskopische Anatomie der nervösen Anteile des Gehörapparates. *Handb. der Neurol. des Ohres.* I. Urban und Schwarzenberg, Berlin, Wien., 1924.

EWALD, I. R.: Physiologische Untersuchungen über das Endorgan des nervus octavus. Wiesbaden, 1892.

FAVILL: The Relationship of Eye Muscles to Semicircular Canal Currents in Rotationally Induced Nystagmus. The Cerebellum VI Research Publication of Association for Research in Nervous and Mental Diseases, p. 530. Williams and Wilkins, 1929.

FERRARO, A., and BARERA, S. E.: The Effect of Lesions of Various Portions of the Vestibular Complex in Macacus Rhesus. *Trans. Amer. Neurol. Assn.*, 64th Annual Meeting, 69, 1938.

FUSE, G.: Beiträge zur Anatomie des Bodens des IV Ventrikels. *Arb. a. d. Hirnanat. Inst. der Univ. Zürich*, 8:213, 1914.

GODLOWSKY, W.: Les centres sous-corticaux du regard et des mouvements associés des globes oculaires. Etude experimentale. Krakow, 1936.

GRAHE: See Leidler.

LE GROS-CLARK, W. E.: Functional Localization in the Thalamus and Hypothalamus. *Jour. Ment. Sci.*, 82:99, 1936.

HELD: Ueber eine direkte akustische Rindenbahn. *Arch. f. Anat. u. Physiol. anat. Abt.*, 257, 1892. Beiträge zur feineren Anatomie des Kleinhirns und des Hirnstammes. *Ibid.*, 435, 1893.

KAPPERS, C. U.; HUBER, G. KARL, and CROSBY, ELIZABETH CAROLINE: The Comparative Anatomy of the Nervous System of Vertebrates, Including Man. Macmillan Co., New York, 1936.

LEIDLER: Experimentelle Untersuchungen über das Endigungsgebiet des nervus vestibularis. *Arb. an d. Wien. Neurol. Inst.*, 21:151, 1916. Das vegetative System und der Vestibularapparat. *Monatsschr. f. Ohrenheilk. u. Laryngo-rhinol.*, 68:513 u. 685, 1934.

LEWANDOWSKY, M.: Untersuchungen ueber die Leitungsbahnen des Truncus cerebri und ihren Zusammenhang mit denen der medulla spinalis und des Cortex cerebri. Jena, Fischer, 1904.

LOEB, JACQUES: Ueber den Anteil des Hörnerven an den nach Gehirnverletzungen auftretenden Zwangsbewegeungen, Zwangslagen u. assoziierten Stellungsänderungen der Bulbi u. Extremitäten. *Arch. f. d. gesamte Physiol.* (Pflüger), 50:66, 1891.

LOEWY, R.: Ueber die Faseranatomie u. Physiologie der formatio vermicularis cerebelli. *Arb. a. d. Neurol. Inst. a. d. Wien. Univ.*, 21:359, 1916.

LORENTE DE NO: Ausgewählte Kapitel aus der vergleichenden Physiologie des Labyrinthes. *Ergebnisse d. Physiol.* (Ascher Spiro), 32:73, 1931.

MAGNUS and DE KLEJN: Experimentelle Physiologie des Vestibularapparates bei Säugetieren mit Ausschluss des Menschen. *Handb. der Neurol. des Ohres.* I. Urban and Schwarzenberg, Berlin-Wien., 1924.

MARBURG, OTTO: Mikroskopisch topographischer Atlas des menschlichen Zentralnervensystems. III. Auflage, Leipzig u. Wien., Franz Deuticke. Entwicklungsgeschichte, makroskopische u. mikroskopische Anatomie des nervus cochlearis, vestibularis u. Kleinhirns. *Handb. der Neurol. des Ohres.* Urban und Schwarzenberg, Berlin-Wien., 1924. Das dorsale Längsbündel von Schuetz, fasciculus periependymalis. *Arb. a. d. Neurol. Inst. d. Wien. Univ.*, 33:135, 1931.

MUSSEN, AUBREY T.: Experimental Investigations of the Cerebellum. The Cerebellum VI Research Publication of Association for Reserach in Nervous and Mental Diseases, p. 381. Williams and Wilkins, 1929.

MUSKENS, L. I. I.: On Tracts and Centres Involved in the Upward and Downward Associated Movements of the Eyes After Experiments in Birds. *Jour. Comp. Anat.*, 50:289, 1930. An Anatomico-physiological Study of the Posterior Longitudinal Bundle in Its Relation to Forced Movements. *Brain*, 36:352, 1913-1914. Das vestibuläre System. Amsterdam N. V. Nord Hollandsche Nitgever maatschapij, 1934.

NAKAMURA, TAMEV: Der Roller'sche Kern. *Arb. a. d. Wien. Neurol. Inst.*, 32:61, 1930.

POETZL, O., and SITTIG, O.: Klinische Befunde mit Hertwig Magendiescher Augenstellung. *Ztschr. f. d. gesamte Neurol. u. Psychiat.*, 95, 1925.

PRICE, I. B., and SPIEGEL, E. A.: Vestibulocerebral Pathways. *Arch. Otolaryngol.*, 26:658, 1937.

RETZIUS, G.: Das Menschenhirn. Stockholm, 1896.

VAN DER SCHUEREN: Etude anatomique du faisceau longitudinal posterieur. *Névraxe*, 13:183, 1912.

SPIEGEL, E. A., and ALEXANDER, A.: Vertigo in Brain Tumors, with Special References to the Results of Labyrinth Examination. *Amer. Arch. Otol.*, 45:979, 1936.

SPIEGEL, E. A., and DEMETRIADES: Beiträge zum Studium des vegetativen Nervensystems. *Pflüger's Arch. f. d. gesamte Physiol.* 196, 1922; 185, 1924; 205-328, 1924. *Monatsschr. f. Ohrenheilk., Laryngo-rhinol.*, 58, 1924.

SPIEGEL, E. A.; OPPENHELMER, M., and PRICE, I.: Higher Integrative Function of the Labyrinth. *Trans. Amer. Neurol. Assn.*, 1937; *Arch. Otolaryngol.*, 26, 1937.

SPIEGEL, E. A., and SCALA, NORMAN P.: The Cortical Innervation of Ocular Movements. *Arch. Ophthalmol.*, 16:967, 1936. Ocular Disturbances Associated with Experimental Lesions of the Mesencephalic Central Gray Matter. With Special Reference to Vertical Ocular Movements. *Arch. Ophthalmol.*, 18:614, 1937.

SAGER, O.: Recherches sur la somatotopic sensitive dans le thalamus des singes. Harlem-de Erven F. Bohm, New York, 1933.

SPITZER, A.: Ein Fall von Tumor am Boden der Rautengrube. *Arb. a. d. Wien. Neurol. Inst.*, 6:1, 1899. Anatomie u. Physiologie der zentralen Bahnen des Vestibularis. *Ibid.*, 25:423, 1924. Ueber die Funktionen der Bogengänge des Ohrlabyrinths. *Monatsschr. f. Ohrenheilk. u. Laryngo-rhinol.*, 59:1, 1925.

TAKAGI, I.: Studien zur vergleichenden Anatomie des Nucleus vestibularis. *Arb. a. d. Wien. Neurol. Inst.*, 27:235, 1925.

WALLENBERG: Die sekundäre Acusticusbahn. *Anat. Anzeig.* 14: 353, 1898; 17:102, 1900.

WINKLER, CORNELIS: Opera Omnia. VII. Harlem-de Erven F. Bolm, 1921.

WHITAKER, T. G., and ALEXANDER, LEO: Der Verbindungen der Vestibulariskerne mit dem Mittel- und Zwischenhirn. *Jour. Psych. u. Neurol.*, 44:253, 1932.

IMMUNOLOGICAL ASPECTS OF BLOOD INVASIONS WITH SPECIAL REFERENCE TO SINUS THROMBOSIS.*

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Some 12 years ago when I joined the Staff of the Mount Sinai Hospital as Bacteriologist, I was greatly stimulated by Dr. Friesner's keen interest and clear conception of sinus thrombosis and tried to adjust the general bacteriological routine to meet the requirements of the otological service.

Generally, I have grown to believe that experimental medicine should always be in close contact with clinical and pathological facts and that a great deal of profit may be derived therefrom.

In this presentation I offer an account of bacteriological and immunological facts in their application to clinical problems, with special reference to sinus thrombosis — that is, rather an account of a bacteriologist's point of view.

In order to follow this plan, it is necessary to give the general immunological background whenever it concerns the clinical problems. In trying to do so, one has to define again a general term used in immunology, which is ordinarily greatly misused and for which many and various interpretations have been given; namely, the term "virulence."

In consideration of this term which is a basis of our understanding of immunology, one usually forgets that virulence does not exist as an absolute entity or as an independent unit. One has to apply the same type of thinking that one uses in any science which deals with changing values.

Virulence, which does not exist independently, simply represents a function of two related variables. One variable is the power to invade the host, and the other variable, which

*Read by invitation at the Seventy-second Annual Meeting of the American Otological Society, Inc., Rye, N. Y., May 22, 1939.

Editors Note: This ms. received in Laryngoscope Office and accepted for publication, May 29, 1939.

is perhaps a little more independent, is the ability of the host to respond to the invasion.

Each of these two variables, in addition, may depend on a long series of their own variables, and, therefore, it is impossible to speak of one variable without any connection with the other. Of course, in studies on specific relationships, one may observe certain constant features, but it is impossible to use these constant features for solution of problems related to other relationship factors.

A simple illustration of such a faulty misconception may be a question, or, rather, a request, tendered by the clinician to a bacteriologist. "I have found," they say, "a certain micro-organism in the patient's urine. Will you please determine whether it is virulent and tell us whether the prognosis is good or bad?"

Now, let us put this micro-organism into a mouse. The mouse succumbs then to a rapidly fatal infection; therefore, the resistance factor may be neglected, and the virulence expressed as an absolute value.

An attempt, however, to transpose these facts or relate them to the behavior of the patient toward the micro-organism invading him, of course, would be a dangerous speculation, because it would exclude completely the consideration of the possible resistance factor of the patient.

Following this line of thinking, Dr. J. L. Goldman and I have analyzed about 168 consecutive blood cultures, positive for streptococcus hemolyticus. In these blood cultures, two obvious groups could be differentiated: First, where the invader was a constant feature; namely, where the invader belonged to the same serological type, and yet where the portal of entry differed vastly.

Second, there was another group of patients where, the portal of entry being of the usual type, the resulting clinical syndrome was conspicuously different from the majority of cases observed. In this manner, then, we had the opportunity to analyze relationship in which the invader was kept constant and the variable factor was the portal of entry; and also vice versa, where the portal of entry was a constant fea-

ture, the strain of the micro-organism apparently representing the variable factor.

In connection with the first, it was interesting to note that a very definite relationship existed between the prognosis and the portal of entry.

TABLE I.

Mortality Rates in Streptococcus Hemolyticus Infections.

Group	Number of Cases	Mortality Percentage
Secondary erysipelas	5	20
Infections of the upper respiratory tract	23	34
Peripheral infections	22	36
Thrombosis of the lateral sinus	43	37
Primary erysipelas	2	50
Gynecological infections	10	60
Osseous and articular infections	8	62
Associated nonbacterial diseases	23	74
Surgical infections	20	85
Acute otitis media with meningitis	4	100
Pulmonary infections	8	100
Total	168	54

Reprinted from the Archives of Surgery, Vol. 34, pp. 82-98, January, 1937. "Streptococcus Hemolyticus Bacteremia. A Study of 168 Cases," by Gregory Shwartzman, M.D., and Joseph L. Goldman, M.D., New York.

Table I gives the mortality rate (in presulfanilamide days) in streptococcus conditions such as skin infections, surgical infections, infections associated with non-bacterial diseases, gynecological infections, osseous and articular infections, etc.

Since the strains were all of the pyogenic group, the wide fluctuations in mortality rate must have been due to the portal of entry. The mortality varied vastly, ranging from between 20 to 100 per cent.

One, therefore, could come to the conclusion that, for instance, the focus of infection in the lungs gave an exceedingly bad prognosis, and that secondary erysipelas complicating a streptococcus infection did not raise the mortality expectation very substantially, etc.

There was, however, one group representing infections of the upper respiratory tract, which was of special interest because the clinical syndrome was markedly different from all other groups. Thus far we have seen 27 cases belonging to this group, as follows:

The patients were admitted to the hospital, usually in early spring or late fall. Most of them were children; none exceeded 20 years of age, many of them were under 10 years of age. They all started their disease with a mild upper respiratory infection which left no trace whatsoever, and there was no tendency toward pus formation. Some time after the primary disease cleared up, the patients developed osteomyelitis of long bones and, infrequently, pus in the joints. These complications were incidental to positive blood cultures, which, however, showed very few organisms and became sterile rather rapidly. The patients did very well, and the operative treatment was beneficial. Thus, in comparison with pulmonary infections (see Table I), the group showed a rather peculiar and different type of body reaction to invasion through the upper respiratory tract, possibly because there existed some difference in the strain of the invading micro-organism.

In special reference to the diagnosis and prognosis of sinus thrombosis, there are certain immunological facts which are of particular interest. The reference, however, requires a preliminary discussion of some laboratory methods which I shall describe without burdening you with too many technical details.

The essential part of the blood culture technique in sinus thrombosis rests upon the fact that a rather large amount of blood is taken, which is cultivated on a variety of rich culture media, and also, that most of the blood is used for seeding into fluid media. Thus, if we take an average of 25 cc. of blood, about 19 cc. is seeded into fluid media, and between 4 cc. and 6 cc. into solid media.

We know that one bacterial cell gives rise to one colony in solid media. Now, if 2 cc. of blood give rise to two colonies, it means theoretically that each cubic centimetre of blood contains one viable bacterial cell. We may, however, expect a certain rate of mortality of bacterial cells in solid media through the sudden change of environment. It seems from experimental estimates that one must have at least five bacterial cells in 2-3 cc. of blood in order to obtain at least one colony in a solid medium blood culture. Therefore, blood seeded into a solid medium and giving rise to one colony must contain initially at least two-three bacterial cells in each cubic centimetre.

On the other hand, the multiplication of bacteria in fluid media is vastly different. One cell is capable of multiplying until it reaches the logarithmic phase where the multiplication goes on in geometric progressions, and within 24 hours, no matter how small the initial invader may be, the final concentration may be about 1,000,000,000 bacterial cells per cubic centimetre of fluid medium. The only difference is in the initial lag period, which lasts between two and six hours, depending on the size of the inoculum. But the moment the logarithmic phase is reached, of course, the initial small difference is wiped out very rapidly, and the final result is the same. Therefore, when one obtains growth in one fluid medium, it may be assumed that there could have been initially present not more than one bacterial cell per 5 cc. of blood of the patient.

These purely quantitative considerations may be of some help in the investigation of facts obtained clinically. For instance, we have convinced ourselves that there is a definite relationship between the number of bacteria recovered from the blood stream and the prognosis in various types of streptococcus hemolyticus infections.

TABLE II.

Relationship of Number of Organisms to Mortality Rates (Prognosis).

Group	Total Cases	Total Mortality Percentage	No. of Fatal Cases	Positive Culture in All Mediums in Fatal Cases	No. of Cases of Recovery	Positive Culture in Fluid Mediums in Cases of Recovery
Infections of the upper respiratory tract	23	34	8	5	15	11
Peripheral infections	22	36	5	5	17	10
Thrombosis of the lateral sinus	43	37	16	9	27	15
Gynecological infections	10	60	6	6	4	4
Associated nonbacterial infections	23	74	17	17	6	4
Pulmonary infections	8	100	8	8		

Reprinted from the Archives of Surgery, Vol. 34, pp. 82-98, January, 1937. "Streptococcus Hemolyticus Bacteremia. A Study of 168 Cases," by Gregory Shwartzman, M.D., and Joseph L. Goldman, M.D., New York.

Table II illustrates an attempt to correlate these two facts where it is seen that the number of micro-organisms is a fairly accurate prognostic indicator.

Now I have come to the statement which I know will meet with some criticism. The statement may be criticized because it is rather dogmatic, and nobody likes dogmas in science, and neither do I. I am thoroughly convinced, however, in the facts about to be presented on the basis of a number of observations over a period of many years. The claim supporting the original observation of Libman is that cases of mastoiditis without any complications have invariably negative blood cultures.

Dr. Goldman recently carried out a large series of blood cultures on cases of noncomplicated mastoiditis immediately following and during operation, thinking that possibly the operative procedure may spread the bacteria into the blood stream. He never found, however, any micro-organisms in the blood stream under these conditions.

On the other hand, one colony, or one bacterial cell, judging from growth only in one flask, means to us already an inflammatory involvement of some vein, and, in particular reference to sinus thrombosis, indicates the involvement of a venous sinus of the head.

Observations on the blood cultures described of this type raise considerably the percentage of agreement between the bacteriological and clinical and pathological diagnosis of sinus thrombosis.

TABLE III.

Blood Cultures in Cases of Thrombosis of the Lateral Sinus.

Preoperative Blood Cultures				Postoperative Blood Cultures			
Fluid Mediums Only				Fluid Mediums Only			
1 Medium*	2 Mediums†	3 Mediums‡	All Mediums	1 Medium*	2 Mediums†	3 Mediums‡	All Mediums
14	4	15	33	2	3	10	17
Total, 66				Total, 32			

*Usually tomato or dextrose broth.
†Usually tomato and dextrose broth.
‡Tomato, dextrose and plain broth.

Reprinted from the Archives of Surgery, Vol. 34, pp. 82-98, January, 1937. "Streptococcus Hemolyticus Bacteremia. A Study of 168 Cases," by Gregory Shwartzman, M.D., and Joseph L. Goldman, M.D., New York.

As you may see, Table III gives an analysis of a group of 66 preoperative and 32 postoperative blood cultures in cases of thrombosis of the lateral sinus, analyzed according to the medium employed. The agreement between the clinical and

the bacteriological diagnosis is very close; *i.e.*, somewhere in the range of 100 per cent or a little below. I think one case was missed.

There is one important fact which may explain to you why the incidence of positive blood cultures in sinus thrombosis is so high; namely, that most of the blood is cultivated in enriched fluid media. Indeed, at least 50 per cent of the positive results were obtained in fluid media only. In other words, if we had used the patients' blood for seeding in solid media only, our incidence of positive blood cultures would immediately drop to 50 per cent, and would drop still more if one used, let us say, one instead of three flasks of fluid media. In the latter case, we would have had only 14 positive results out of 66 blood cultures, instead of 65 out of 66.

The almost perfect agreement of results of blood cultures with the clinical diagnosis convinces us that it is invariably necessary to consider the presence of the streptococcus hemolyticus in the blood stream as an indicator of some pathological involvement of a blood vessel.

This being the case with streptococcus hemolyticus, it should not, however, be generalized for other types of bacterial invasion, as, for instance, we pay no attention to a single fluid medium blood culture positive for streptococcus viridans. On the contrary, very frequently one observes a transitory and innocuous invasion of the blood stream with a rapid disappearance of the streptococcus viridans without consequence.

This type of reasoning, incidentally, may be also somewhat helpful in the differentiation of inflammatory reactions of the meninges from brain abscesses.

On many occasions, we may see pus cells in the spinal fluid, accompanied by only a few bacterial cells. In most cases, we feel that this picture indicates a brain abscess rather than a diffused meningitis, provided, of course, that a complete block is ruled out. That has been borne out very frequently by clinical observations. A large number of pus cells is usually paralleled in meningitis by a considerable number of bacterial cells in the spinal fluid.

It is of great interest whether the streptococcus hemolyticus is the only micro-organism which is capable of blood invasion

following infection of the middle ear and the mastoid. After all, there are many other micro-organisms present in the original focus which may get into the blood stream if they were given a chance by the body.

I have compiled figures of the bacteriology of the mastoid and the middle ear obtained in our hospital during the past five years, as follows:

TABLE IV.

Bacteriology of the Mastoid and the Middle Ear During a Five-Year Period (1934-1939).

Number of Cultures		
Mastoid	Middle Ear	Micro-organism
261	98	Streptococcus hemolyticus, pure
36	113	Streptococcus hemolyticus in association with B. proteus, B. coli, staphylococcus, B. pyocyaneus, diphtheroids
10	88	Staphylococcus, pure
10	28	Staphylococcus in combination with B. coli, pyocyaneus, diphtheroids
6 — Type I	11 — Type I	Pneumococcus
1 — Type II	3 — Type II	Pneumococcus
44 — Type III	22 — Type III	Pneumococcus
12 — Types V, VIII, XIV, XVIII	25 — Types V, VII, VIII, XI, XII, XIV, XVI, XVIII, XX, XXIII, XVIII	Pneumococcus

In Table IV, the number of cases cultured is summarized with an indication whether there was a pure culture of streptococcus hemolyticus, whether streptococcus hemolyticus was associated with a number of various other micro-organisms, whether there was a pure culture of staphylococcus, whether the staphylococcus was in combination with the other micro-organisms, and also an indication is given of the various types of pneumococcus encountered.

One very striking fact is shown in Table IV; *i.e.*, pure cultures of streptococcus hemolyticus were mostly predominant in cases of mastoiditis; streptococcus hemolyticus in mixture with other micro-organisms being seen in a rather insignifi-

cant group. The low incidence of the pneumococcus in cases of mastoiditis is also of interest.

On the other hand, pure cultures of the streptococcus hemolyticus in the middle ear were less frequent. Contaminations or associations with other bacteria were rather high, and the number of various types of pneumococci also was much larger than in mastoiditis. This, of course, can be very simply explained by the fact that the middle ear communicates by means of the Eustachian tube with the upper respiratory tract, while the mastoid represents a secondary line of defense.

Thus, when the second and third lines of defense are approached, micro-organisms other than streptococcus hemolyticus are gradually eliminated and the streptococcus hemolyticus gets the upper hand.

On the cases of the Otological Service already examined in Table I, we have taken a total of 162 blood cultures during the period of five years indicated (excluding repetitions); and obtained 34 positive blood cultures. Out of the latter, 29 showed streptococcus hemolyticus, and the remaining only contained pneumococcus and staphylococcus aureus (see Table V). Three of the latter cases, however, were not primary sinus thrombosis.

TABLE V.

Blood Cultures — Otological Service — Five-Year Period (1934-1939).

Total No.	No. of Positive Cultures	Strep. Hem.	Micro-organism Pneumococcus			Staph.
			III	XIV	XVIII	
152 (excluding repetition)	34	29	1	1	1	2

Now, coming back to the immunological interpretation of the various clinical processes, one has to refer to certain problems which have been successfully investigated in the past few years.

In the first place, Lancefield and Heidelberger classified the streptococcus hemolyticus, on the basis of specific carbohydrates. The work resulted in an entirely new grouping, in which it was found that the streptococcus hem-

olyticus can be subdivided into Groups A, B, C, D, E, F and G. Among these groups, only Group A is pathogenic to man. The remaining groups are of animal origin, although they may be carried by man but are incapable of producing human pathological lesions. As a matter of fact, in our personal experience we have never found a non-A group responsible for any significant human lesion, and we have certainly not found a single one responsible for any inflammation in the head.

Now, strangely, Group A, outside of being pathogenic for man, is also endowed with one extremely interesting property which has recently received a great deal of attention of various investigators. The observation deals with so-called fibrinolysis, discovered by Tillett and Garner in the Rockefeller Institute in 1933. They started out with the basic idea that the effect of streptococcus hemolyticus upon the fibrin must be peculiar, because of the observation that the exudates derived from serous cavities infected with streptococcus hemolyticus are devoid of fibrin and appear extremely thin.

Their investigations showed that the streptococcus hemolyticus, which later was identified as the "A" type, produces a substance actively interfering with the transformation of the fibrinogen into fibrin, and is actually capable of producing complete fibrinolysis when grown in human blood.

The remarkable part about this is that the fibrinolysin, which, incidentally, is not an enzyme, is of such specificity that it produces no effect whatever upon any other fibrin except human and, therefore, it may be considered of importance in propagation and dissemination of disease. Moreover, the workers found that human fibrin itself acquires an active immunity in the course of an infection and that eventually, as the disease progresses, the fibrin becomes so thoroughly immune to this fibrinolysins that no fibrinolysis can be obtained *in vitro* and *in vivo*. As a matter of fact, the quantitative estimation of the antifibrinolytic properties of the patient's fibrin during the progress of the disease may even serve as a useful prognostic indicator; the recovery from infection being associated with resistance of the fibrin to fibrinolysins in high titre.

Thus, there seems to be a rather startling and interesting observation concerning the type of streptococcus hemolyticus

responsible for sinus thrombosis. An attempt will be made a little later to determine what rôle the fibrinolysis may play in the disease.

Moreover, streptococcus hemolyticus of certain types produces certain factors which are called the "Reynals spreading factors" and have the peculiar ability of enhancing the permeability of the tissues with which they come in contact. This effect is usually estimated by the rate and intensity of diffusion of India ink injected into the skin. Where the India ink is mixed with the spreading factors, the spreading of the dye becomes extremely rapid. Thus, the streptococcus hemolyticus differs from many other micro-organisms in the two important features, *i.e.*, spreading power and fibrinolysis.

Of course, in the considerations of blood invasion by streptococcus hemolyticus, one has to differentiate a number of various phases. At each phase there is an opportunity being offered for the establishment of an equilibrium between the invader and the host. Inasmuch as the initial infection of the middle ear or mastoid may result either in involvement of lateral sinus and blood invasion, or remain localized and eventually disappear, this transitional state must in reality consist of a number of intermediate immunological phases.

In the first place, one may assume (this is supported by some experimental work) that when an infection establishes itself, the bacterial toxic factors diffuse into the surrounding tissues some time before the bacterial cells begin to spread into the surrounding tissues.

At the bacteria-free stage there may already occur pathological changes in the wall of the lateral sinus, as roughening of the intima, leading to formation of sterile thrombi, which I believe in many cases precede bacterial invasion.

It is very interesting that the primary thrombi apparently produced exclusively by the bacterial toxic factors and having nothing to do with the bacteria themselves are different histologically from those of so-called secondary bulb thrombosis, as so well shown by Dr. Friesner and Dr. Druss.

The inflammatory reaction seen in the vein in the neighborhood of the thrombus is seemingly produced, however, by the

effect of the bacteria themselves after they spread to the thrombosed vein. This represents the second stage of the process. The following stages may be, then, as follows:

Invasion of the thrombus by the bacteria; multiplication of the bacteria in the thrombus; and, finally, either destruction of bacteria in the thrombus due to the formation of the fibrin and thrombus organization; or, survival of bacteria with destruction of the thrombus and dissemination of the infected thrombotic particles by means of the circulation. Here it should be emphasized that the micro-organisms which have the means to dissolve the fibrin and thus prevent the organization of the thrombus are those which have the best chance to disseminate the infection and invade the blood stream.

Now, returning to the fact that the streptococcus hemolyticus is the only micro-organism which possesses strong and specific fibrinolytic agents, it becomes obvious why the streptococcus hemolyticus is practically the only micro-organism which invades the lateral sinus following mastoiditis.

Since the breaking down of the primary thrombus is associated with the superimposed bacterial infection of the thrombus, secondary thrombi which are set up by these infected particles of fibrin at the later stage of the disease, according to the pathological observations and according to our experiments and our thinking, obviously may be of severer type, and may show also incidental phlebitis and more pronounced histological damage leading to necrosis, as observed by Dr. Friesner and Dr. Druss in secondary bulb thrombosis.

Another point which was always a mystery in my mind is how a ligation of a sinus helps to cure the disease. As a matter of fact, if you close up a pouch filled with pus, somewhere in the abdomen, you certainly do a lot of harm to the patient. Here you are actually closing up an infected focus instead of allowing it to drain. The inflammatory reactions of the remaining parts of the infected vein certainly never sterilize the infected focus. There always must be some micro-organisms left at the site after the operation, even following vein excision.

Apparently the favorable effect of the operative interference is not due to a successful sterilization but to the limita-

tion of the process and prevention of secondary thrombi which according to our conception are more harmful than the primary thrombosis. In addition, as already pointed out, later in the disease the fibrin eventually becomes immune to the dissolving effect of streptococcus hemolyticus and thereupon may help to destroy the bacterial invader through organization of the primary thrombus as well.

There is also another aspect in connection with this which it would be impossible to describe in this presentation, but which seems to indicate very clearly the important rôle which the portal of entry or the route of dissemination of bacteria in the body plays in determining the severity of the infection. My work on the phenomenon of local tissue reactivity shows that the hematogenous route is capable of producing extremely severe damage, while the local infections, or reinfections, induced by direct spreading are definitely milder and result in prompt recovery.

In conclusion, I would like to point out that the bacteriologists may greatly benefit by elaborating the analysis of immunological aspects of sinus thrombosis, since this disease has been already so thoroughly investigated by the otologists and pathologists.

THE USE OF THE MONOCHORD IN ROUTINE TESTS OF HEARING.*

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The purpose of this paper is to endeavor to answer the question, "Is the routine use of the monochord worth while?" It is frankly expected that the ensuing discussion will be of far greater value than the arguments we have to offer. We have used the monochord routinely during the past 18 years and have felt that it gave us information of much value. At the same time, it seems that few clinicians employ this instrument. Have we been attaching too much importance to its use, and are its findings merely reduplications of those of other and more useful instruments? Is it of value only in research work, and an affectation in clinical practice? Or does it really add something to the functional hearing test which might be employed routinely to advantage?

The original monochord was an instrument used by the ancient Greeks for tuning purposes and to measure the scale arithmetically. Struycken adopted the general plan of this instrument, modifying it to its present form, in an endeavor to overcome the inaccuracies of the Galton whistle, used in testing the upper tonal range.

We are largely indebted to Bunch[1] for calling attention to the place of the monochord in our present-day conception of hearing tests. His numerous publications prove conclusively its value in research work and strongly suggest its usefulness in clinical practice. He found a higher correlation with other clinical tests by the monochord than by other instruments, such as the whistles, Koenig cylinders, used in testing the upper limits. In a personal communication Bunch[2] says, "I would say that of all the devices available for determining the upper limit, I still consider the monochord the most satisfactory for both bone conduction and air conduction. The

*Read at the Seventy-second Annual Meeting of the American Otological Society, Rye, N. Y., May 23, 1939.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 29, 1939.

chief handicap in its construction is the impossibility of measuring the intensities of its tones."

With the monochord one can compare the upper limit of hearing by air conduction and by bone conduction, as it has practically the same amplitude for both. This is in contradistinction to the tuning fork, the stem of which, used for bone conduction, has only about one-hundredth the amplitude of that of the prongs.[3] Also, there is no appreciable dampening with the monochord. Normally, the monochord is heard by bone conduction from 1,000 to 2,000 d.v. higher than by air conduction, suggesting that hearing by bone conduction is actually better than by air conduction.

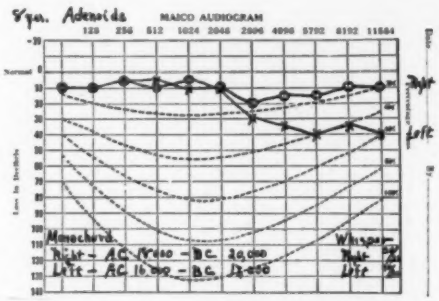

Fig. 1. Age 8 years. Adenoids. Marked drop in upper tonal range on left, referred to by Crowe. In addition, monochord shows a lowering of upper tonal range on right.

The higher tones of the audiometer, even at the maximum intensity, are of much less intensity than those of the monochord.[4] While the audiometer gives us quantitative determinations of the threshold of hearing, both for air and bone conduction, those commonly in use in clinical practice do not extend above 8,192 d.v. or 11,584 d.v. Many observers have expressed dissatisfaction with the bone conduction findings in the upper tonal range. Shambaugh[5] states that, with the bone conduction receiver, the higher frequencies of the audiometer are heard increasingly by air, and that the bone conduction curve for the upper half of the tonal range is unreliable.

Of course, it is rather arbitrary to state that any given frequency is the upper limit of audibility. Actually, one can

only say that any obtained measurement is the upper limit for that instrument employed in testing. Bunch[4] states that it is probable that what may be considered an actual deafness for these high tones is but a loss in sensitivity. Higher tones might be heard if it were possible to produce tones of greater intensity than is possible with the monochord. But as the monochord has the greatest intensity of the instruments commonly available and is most satisfactory for bone conduction, it would seem that it might be taken as a standard for testing the upper limits.

We realize that there is a loss for high tones commonly seen as an accompaniment of advancing age. This has been care-

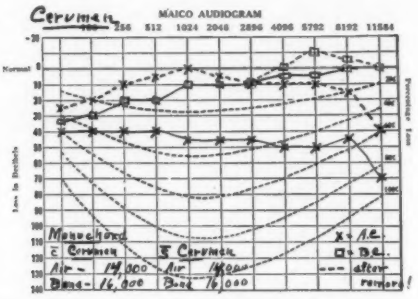

Fig. 2. Audiogram of case of impacted cerumen — purposely tested before removal. Marked increase in hearing by air conduction but no change with monochord noted following removal.

fully studied by Bunch and Raiford,[6] and Ciocco,[7] who found that there was a gradual drop for frequencies above 2,048 with each decade above 20. With the monochord, we may expect a lowering of the upper limit by 1,000-2,000 d.v. for each such decade, taking 20,000 to 22,000 as the average normal for a person in the early twenties.

This, of course, will show some variation, according to the past medical history of the patient. Various toxemias may definitely affect the acuity of hearing for high tones. A previous otitis media may cause a lowering of the upper limit, which cannot be attributed simply to mechanical interference, from residual pathology in the middle ear. Were this so, we should expect to find a greater difference than the normal

spread between air and bone conduction with the monochord in those having had middle ear disease. Actually, we find the ratio about the same. We also know that no differences are found in the determination of the upper limits by the monochord before and after removal of impacted plugs of cerumen, or after incision of the membrana tympani. Consequently, we may feel justified in saying that middle ear lesions

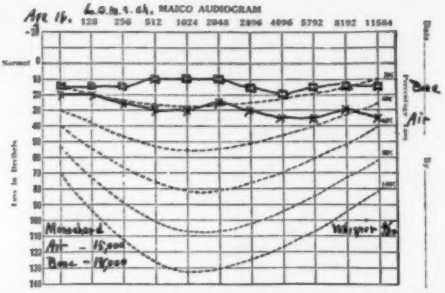

Fig. 3. Age 16 years. Chronic suppurative otitis media. Fair response to monochord. Better prognosis for hearing than following case. No increase in spread between air and bone conduction with monochord.

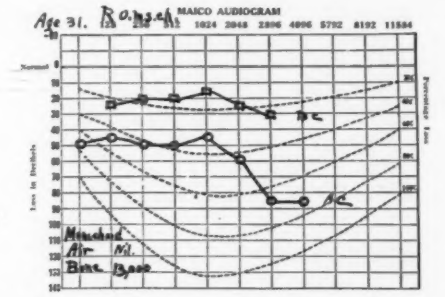

Fig. 4. Age 31 years. Chronic suppurative otitis media. Poor response to monochord. Marked spread between air and bone conduction.

definitely produce certain degenerative processes in the internal ear, and the effect of these processes may become manifest in the readings obtained on the monochord.

We have long recognized that all forms of perceptive type deafness exhibit a loss for high tones before showing impairment of the lower tonal range. Crowe's[8] studies show that in

partial or complete obstruction of the Eustachian tube from nasopharyngeal pathology, or with a lesion interfering with the movements of the malleus and incus, hearing impairment begins with the higher frequencies. Similarly, an acoustic tumor or Ménière's disease may show upper tone loss before the rest of the tonal range is affected.

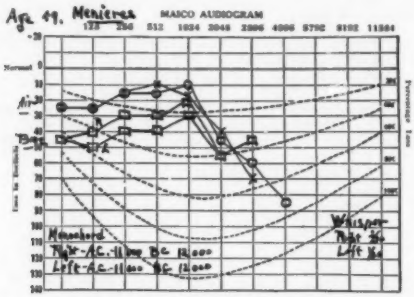

Fig. 5. Age 59 years. Ménière's disease. Symptoms controlled by Furstenburg treatment. Audiogram alone would suggest complete loss of high tones. This is contradicted by monochord.

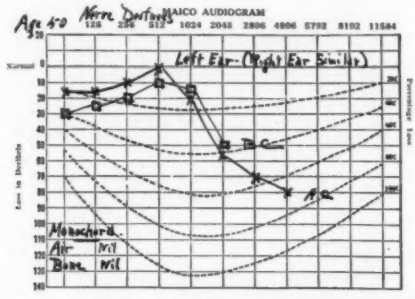

Fig. 6. Age 50 years. Nerve deafness. Definite loss of upper tones — at least as produced by monochord. Conversation heard at 18 feet.

The value of the monochord becomes more apparent when we consider that the determination of the upper limit of audibility by bone conduction is the most accurate indication of the condition of the nerve. According to Dean and Bunch,[9] lowering of the upper limit by bone conduction is more indicative of a lesion of the nerve or end-organ than lowering by air conduction.

It is generally agreed that the audiometer alone is not sufficient to make a differential diagnosis of the different forms of deafness but must be supplemented by the various tests with the tuning forks. Oftentimes the use of the monochord may materially change even this audiometric picture and give us quite a different conception of the case. Bone

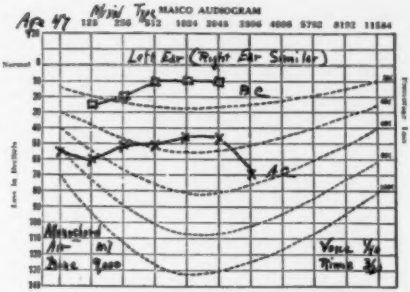

Fig. 7. Age 47 years. Marked deafness, probably "mixed type." Monochord shows there is response by bone conduction above what is shown in the audiogram. Case seems suitable for bone conduction hearing aid.

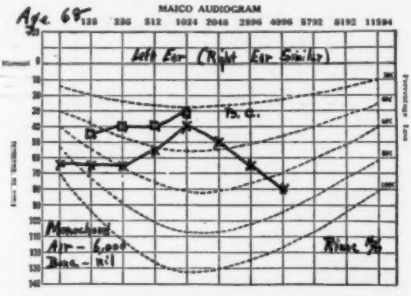

Fig. 8. Age 68 years. Nerve deafness. Poor bone conduction and no response by bone to monochord. Marked senile type. Would probably do better with speaking tube.

conduction with the audiometer may be lost for frequencies of 1,024 and above, and yet be heard up to 15,000 d.v. by bone conduction with the monochord. In another case, this same limit of bone conduction may be found with the audiometer and yet no response be obtained with the monochord. Taking the audiometric readings alone, these might present similar pictures, yet they are far different as regards the status of

the internal ear. Certainly, in the former case one could not be justified in saying that there was a very marked nerve deafness involving the upper limit, where it is possible to hear by bone conduction up to 15,000 d.v. on the monochord. In the second case, however, one might feel justified in assuming a much more severe degree of nerve involvement. Again, we

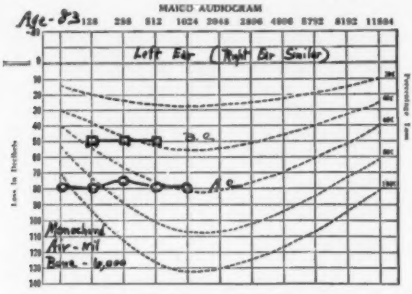

Fig. 9. Age 83 years; yet probably less senile than preceding cases.

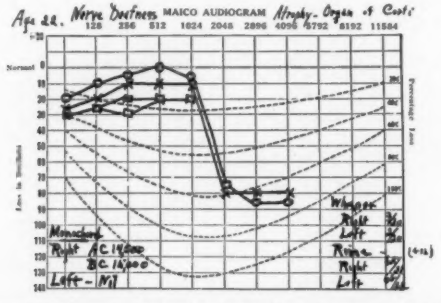

Fig. 10. Nerve deafness — probably atrophy of organ of Corti. Audiogram typical of Crowe's cases. Probable etiology — mumps. Audiogram alone would suggest symmetrical loss of high tones but this is contradicted by the monochord, which is not heard by either air or bone conduction on the left. Labyrinth tests show lack of normal response from left labyrinth.

may obtain fairly normal bone conduction readings with the audiometer and yet find a significant drop in bone conduction with the monochord. Cases which show lowering of the upper limit by air conduction may have fairly normal upper limits by bone conduction, which would seem to eliminate destruction of the nerve mechanism for perception of these tones. We may see cases where all bone conduction, both by audiom-

eter and monochord, is lost. In such cases, one would feel justified in assuming the most severe forms of perceptive deafness. These are typical of the extreme senile type, and may explain the notoriously poor bone conduction commonly seen in the aged. Of course, the ear not being tested must always be carefully masked.

In cases of chronic suppurative otitis media, we generally find a lowering for the whole tonal range on the audiometer. We are apt, however, to note considerable variation in the readings obtained with the monochord. And this may be of some prognostic importance as regards the conservation of hearing. With a normal or relatively normal reading on the monochord, especially by bone conduction, we feel that there is a rather good prognosis as regards hearing; while with a

M.B.

Rotation

Rotated to left, H.Nyst. to R.- 30"
" " right, " " " L.- 15"

Caloric - 62° F.

Right - R. Nyst. to L. - 30"
Left - No Nyst. - 3' 30"

Fig. 11.

relative low reading on the monochord, the outlook for hearing may be quite poor. Of course, this is only relative and is of far less importance than the clinical picture of the middle ear. Yet, one cannot help feeling that an index of the condition of the internal ear, as manifested by the determination of the upper limit by both air and bone conduction, is of considerable importance in a prognostic way.

It would seem, therefore, that some such device as the monochord, which is reliable in a qualitative sense at least, could be employed to advantage in testing the upper limit of hearing.

CONCLUSIONS.

1. Accurate determinations of the upper limit of hearing, especially by bone conduction, give the most reliable informa-

tion regarding the status of the internal ear and may be of prognostic importance in regards to hearing.

2. The monochord is the best available device for measuring both air and bone conduction in the upper tonal range; granting the handicap of its being a qualitative instrument.

3. While no single instrument or test is sufficient alone to determine the differential diagnosis of different types of deafness, the monochord can be used routinely to advantage with the tuning forks and the audiometer in the functional tests of hearing.

REFERENCES.

1. BUNCH, C. C.: Functional Hearing Tests in Normal Cases. *Ann. Otol., Rhinol. and Laryngol.*, 33:1-174, March, 1924.

2. BUNCH, C. C.: Personal Communication.

3. SONNENSCHEIN, R.: The Rationale of Tuning Fork Tests. *Ann. Otol., Rhinol. and Laryngol.*, 37:1-309, March, 1928.

4. BUNCH, C. C.: The Monochord in Upper Limit Determinations as an Adjunct to Audiometry. *Ann. Otol., Rhinol. and Laryngol.*, 37:1-372, March, 1928.

5. SHAMBAUGH, G. E., JR.: The Interpretation of Bone Conduction and Other Tests of Hearing. *Ann. Otol., Rhinol. and Laryngol.*, 35:3-779, Sept., 1936.

6. BUNCH, C. C., and RAIFORD, T. S.: Race and Sex Variations in Auditory Acuity. *Arch. Otolaryngol.*, 13:3:422, March, 1931.

7. CIOCCO, A.: Observations on the Hearing of 1980 Individuals: A Biometric Study. THE LARYNGOSCOPE, 42:11-837, Nov., 1932.

8. CROWE, S. J.: Diagnosis and Differential Diagnosis of Deafness. *Arch. Otolaryngol.*, 28:5-663, Nov., 1938.

9. DEAN, L. W., and BUNCH, C. C.: The Study of the Tonal Ranges in Lesions of the Middle Ear. *Ann. Otol., Rhinol. and Laryngol.*, 31:3-617. Sept., 1922.

Professional Building.

DISCUSSION.

DR. HAROLD WALKER (Boston): I enjoyed Dr. Hill's paper very much, especially his slides, which I think were very impressive, showing the advantages to be obtained by using the monochord.

The subject of testing by bone conduction has always been an important one, and any information which will help us in measuring the hearing by bone conduction is valuable. Dr. Hill's paper on the virtues of the monochord is very timely. A good deal of work has been done recently upon different methods for obtaining an accurate index of the efficiency of the internal ear. I refer particularly to the work of Dr. Fowler, of New York, and of Dr. Ernest Barany, of Upsala.

A good many years ago, I became interested in the monochord, and finally purchased one and used it with a great deal of satisfaction until the wire broke and the measuring device was lost. About that time, the audiometer was invented, and I am sorry to say that I had almost forgotten how valuable an instrument the monochord is. As Dr. Hill states in his paper, it is the only method by which the highest tones can be detected by bone conduction, and while it is not possible to determine the quantitative value, it is possible to gain a fairly accurate idea of the condition of the internal ear. There are cases, especially of the senile type, where it is not possible to obtain any evidence of the efficiency of the internal ear by means of tuning forks or the audiometer, and yet these cases may possess fair hearing for the higher tones.

If the operation of the fenestration of the external semicircular canal proves to be of value, it will be necessary that one be able to obtain an accurate index of the efficiency of the internal ear. I believe the monochord may be of great value in determining the advisability of such an operation.

I am glad that Dr. Hill has been willing to describe in detail the usefulness of this instrument in his practice, and I heartily agree with him in his conclusions.

DR. C. C. BUNCH (St. Louis): There is very little that I can add to what Dr. Hill has said. In regard to the intensity measurements which I found to be the chief handicap, I just wish to give you a little experience, the result of an experiment which we have tried this last year showing the difficulties in measuring the intensity of sound from the monochord. Two of our graduate students start walking down the corridor in our clinic room, which is almost the length of this room. When at the end of the clinic room, in spite of all the noises in the room, conversation, fans, dishes rattling, etc., they still could hear the monochord. One of them reported that he thought he could walk down the corridor to the right as far as the elevators and still be able to hear it. The sound is what we would call a "tiny" sound, yet it has very great intensity.

The inability to control the intensity for measurement is, I think, the handicap which one must overcome in order to use the monochord effectively. This, of course, can be done better with electrical means as with the audiometer, but, unfortunately, the audiometer does not produce tones to the upper limit. The 1A audiometer which we use goes to 16,384 d.v. at an intensity of 40 units, which is not very loud, and many of us who are older than 30 years do not hear this tone at the maximum which can be given, which, by the way, is far from the threshold of pain.

Whenever we use the monochord in the soundproof room, we always turn on the fan, because the masking noise of the fan does not interfere with the hearing of the monochord in any way that I have ever been able to determine.

There are three methods that are ordinarily available for determining the upper limit: The monochord, as you have stated, the Galton whistle and the Koenig bars.

With the monochord, the tone is made by stroking the wire with a little sponge which is soaked in carbon tetrachloride or some other solution. It is very easy to touch your finger to the wire and deaden the high pitched sound to which the patient is listening. You can then ask him to tell whether he hears the sound of the squeak or that of the rubbing. If he is unable to tell the difference, you know that you are reaching the upper limit. This is quite important in dealing with clinical patients who may not always have normal intelligence.

With the Galton whistle, you may have confusion because the patient will hear the hiss of the whistle in addition to the sound of the wind, and it is impossible to shut off the wind sound without cutting down the hiss of the whistle. It is difficult to get the patient to respond only to the whistle.

With the Koenig bars, the noise comes as the result of striking the bar with a hammer. Your patient should respond to the ring rather than to the thud of the hammer, which may also be difficult for clinical patients.

One thing which has always bothered me is the inability to mask the opposite ear when you are doing bone conduction tests with the monochord. I have inserted two Barany buzzers in the ears of a patient and put the monochord against his head and found that he could often tell when it was squeaking, regardless of the fact that the ears were masked with Barany buzzers. I was noticing in Dr. Hill's records that he gave the high tone for one ear alone and I would like to ask him how he succeeded in masking the sound from the opposite ear.

We found the monochord, as I have stated before, the most satisfactory of the three instruments that are available for measuring the upper tone limit. Thank you!

DR. WERNER MUELLER (Boston): It pleases me to have Dr. Hill come forward to champion the monochord. I have always loved the monochord as a musical as well as a scientific and clinical instrument. Musically, it has been of the greatest importance as a way station between the bowstring of our primitive ancestors and the modern grand piano. Scientifically, it has no peer as a producer of accurately measurable tones. As an audiometric instrument, however, it has never quite occupied the place it deserved, even in the days before the audiometer. The reason for this has always been a riddle to me. The monochord is simple in construction and manipulation.

I brought my instrument along because I thought it might help to illustrate some of the points. The monochord consists essentially of a steel ruler, on which a steel wire is stretched, and the length of the vibrating portion of the steel wire can be adjusted by a small sliding metal block. The instrument is easily set. It is now set at 6,000 vibrations. There are numbers engraved on the narrow side of the rule, each representing thousands of cycles. Here is 6,000; I hope you can hear it.

Its tones are pure, the pitches are accurate and easily adjusted. The tones are free from adventitious noises of any consequence if carbon tetrachloride or benzine are used.

It can be used for bone conduction by merely placing the beak end of the monochord against the patient's mastoid. The monochord is usually provided with a small button made of composition (this happens to be hard rubber) in order to make the pressure less painful to the patient. I always ask the patient to lean his head against my monochord with comfortable pressure.

While I have the instrument in my hand, I might also illustrate a point Dr. Bunch brought out. I learned that from him last week in St. Louis, and it is a very valuable thing to know. You hear the tone. Now I touch the wire lightly with my thumb and the tone disappears. If there is an adventitious noise, the patient can easily spot it. You can't do that with the Galton whistle.

It lacks the distressing adventitious noises produced by blowing the Galton whistle or striking the Koenig cylinder. It can be used for bone conduction. If kept free from rust, it will retain its purity and accuracy of tone almost indefinitely. If the string should break, it could easily be replaced by any moderately skillful mechanic.

I might say the size of the string makes little difference, nor the tension. The pitch of the monochord string is determined by practically only one factor and that is the speed with which sound will travel in steel.

The adjustments to the various pitches are far simpler than those necessary for the use of the Galton-Edelmann whistle. You know, that always requires

two settings, and usually when you get one set and you pick up the instrument, you twist it the least little bit and the setting is out.

No table need be consulted and there is no rubber bulb to wear out. The pitch range of the monochord was originally from 6,000 cycles per second up. You noticed in one of Dr. Hill's audiograms there was no response to the monochord at all because the monochord as used by longitudinal vibrations goes down to only 6,000. By the aid of Schaefer's modification, which involves the use of a bow and hammer for exciting the string to transverse vibrations, you can carry the tones down to 435. In this way, upper limits that lie below 6,000 cycles can be determined.

If there is any reason at all for determining the upper limit of tone perception, there certainly is a place for the monochord in the equipment of the otologist.

When you get back home, look for your monochord and, if you can find it, take it out of its case and play with it. It will be worth your while.

DR. FREDERICK T. HILL (Waterville, Me.): I said in my written paper that I felt that the discussion would be more valuable than the paper, and I think that is confirmed. I was indebted to Dr. Walker for calling my attention some years ago to the monochord. I am sorry he has broken the string, but I know he will get that fixed.

Dr. Bunch brought out the very important point of differentiating the rubbing sound from the true tone. I think that is very valuable. I will agree with him on the difficulty of masking. In the case under discussion, the other ear apparently was completely deaf for the high tones. At least we felt this was confirmed by the labyrinth tests, so I don't believe masking was such an important factor.

SKULL FRACTURES INVOLVING THE EAR. A CLINICAL STUDY OF 211 CASES.*

DR. W. E. GROVE, Milwaukee.

The material from which this survey is drawn consists of two series of cases. The first concerns 211 cases of skull fracture selected from 1,187 cases of head injury which I have examined in the last 13 years. The criteria on which the diagnosis of skull fracture was based were bleeding from the ear, positive X-ray evidence of fracture, or a complete loss of ear function. As I shall show later, it is possible that a few of the cases which bled from the ear were not skull fractures, and it is also possible that in some the total loss of function, or cochleovestibular paralysis, was not caused by a fracture, but these exceptions must be very few in number. On the other hand, it is also possible that there were more than 211 cases of skull fracture in the material studied because X-ray data was either inaccurate or not available.

Inasmuch as very few of these cases were seen until after the period of hospitalization and, therefore, gave me no information of the immediate effects of head trauma and skull fractures, I examined another series of 152 consecutive head injuries within 24 hours of their admission to the Emergency Unit of the Milwaukee County Hospital between May 17, 1938. and July 24, 1938. In this series there was some evidence of skull fracture in 17 cases, with two deaths, or a mortality of 11.7 per cent.

My main object in examining the last series of freshly injured head trauma cases was to ascertain in how large a percentage of the 150 cases which survived, any ear findings were present, and I found that some ear sign or symptom was present in 49 cases, or 32.66 per cent. This figure is very similar to that of Mygind,[1] who by otoscopic examination of 142 cases found traumatic changes in about 50 per cent. He admits, however, that in about one-half of this 50 per cent the changes consisted simply of hyperemia of the tympanic

*Read at the Seventy-second Annual Meeting of the American Otological Society, Rye, N. Y., May 23, 1939.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 29, 1939.

membrane, which soon disappeared. Discounting these evanescent changes, which, to my mind, are very difficult of interpretation, his percentage is reduced to 25 per cent. Mygind[1] found signs of vestibular trauma in 33.3 per cent of his cases as compared to 26.6 per cent in my series. The signs noted in my series of 150 cases were: vertigo associated with spontaneous nystagmus, 40 times; fracture of the external canal, six times; a ruptured drum and bleeding from the ear, twice; a ruptured drum and escape of cerebrospinal fluid, once. The 17 cases of skull fracture consisted of six vault fractures, five basal skull fractures and six fractures of the external canal. The fractures of the external canal may have been a manifestation of longitudinal fractures.

For the purposes of study and analysis, I divided my first series of 211 cases, which were largely medicolegal in character and which were not examined until after the period of hospitalization, into: *a.* fractures of the vault alone, or of the vault in combination with other parts of the skull, exclusive of the temporal bone; and *b.* temporal bone fractures.

a. The vault and miscellaneous skull fracture cases studied were: Pure vault fractures, 34; vault fractures running into the base, seven; vault fractures associated with fractures of the paranasal sinuses, one; anterior fossa fractures, two; fractures of the base other than petrous fractures, two; fractures of the base and face, two; and fracture of the frontal sinus, one; in all, 49 cases.

b. The temporal bone fractures were subdivided into longitudinal fractures and transverse fractures of the temporal bone. There were 146 cases of longitudinal fracture of the temporal bone, of which 65 were on the right side and 46 on the left side. Bilateral bleeding occurred in 34 cases. At least 12 of these were associated with fracture of other parts of the skull; in one of the unilateral cases I was unable to determine which ear had bled.

There were 16 cases of unilateral cochleovestibular paralyses which I have, for the purpose of discussion, classified as transverse fractures of the pyramid. Of these, eight were classified as pure transverse fractures of the pyramid. Seven of the remaining eight were combined with unilateral longitudinal fractures as evidenced by bleeding from the ear, and

one case was combined with bilateral longitudinal fractures of the petrous bone. As will appear below, the absolute diagnosis of transverse fracture of the pyramid cannot be made with certainty in all of these cases with the exception of those verified by X-ray, for fracture of the labyrinthine capsule is not the only traumatic pathology which can explain the cochleovestibular paralysis, although it is the most probable.

ETIOLOGY OF SKULL FRACTURES.

All of the skull fractures in the present survey were caused by blunt force. Gunshot wounds are not included. As I[2] pointed out in 1928, all such injuries are caused by some broadly acting force which has a wide area of impact against the skull. Many are due to automobile accidents, and the remainder occur in the industries. The head is struck by falling objects; the individual falls from a height; is thrown from an automobile, bicycle, motorcylcle or horse, striking his head against some solid object; or the head is jammed between two solid objects, one of which is in motion. While the force is usually propagated to the head by way of the vault or the face, it can also reach the head from a jar on the heels or a fall on the buttocks (Voss[3]), being transmitted via the vertebral column to the occipital bone and through it to the petrous pyramid. Furthermore, when force is applied to those bones of the face which are directly adjacent to the temporal bone, such as mandible and zygomatic process, damage to the inner ear can easily occur. Voss also feels that the place of predilection for the production of serious injuries, especially pyramidal fractures, is the occipital or occipitomastoid region.

Mellenger[4] states that all fracture lines except those through the posterior fossa tend toward the sphenoid, and if the force is not spent it is deflected by the solid basisphenoid, to continue along the anterior border of the pyramid. Rawlings[5] enumerates the usual sites of impact as: *1.* Midfrontal region; *2.* lateral frontal region; *3.* external ear; *4.* mastoid region; and *5.* occipital region. He presents an excellent diagram illustrating the direction taken by fractures resulting from force applied to these head regions (see Fig. 1).

Mellenger[4] further points out that the temporal bone enters into the formation of the side and base of the skull; it forms

two-thirds of the floor of the middle, and one-third of the floor of the posterior fossa. The sutures connecting it to the neighboring bones tend to remain open with fibrous gaps. Thus, a certain amount of movement or give from below is provided for. If a blow on the chin is of sufficient force, the whole temporal bone may be displaced upward or the condyle

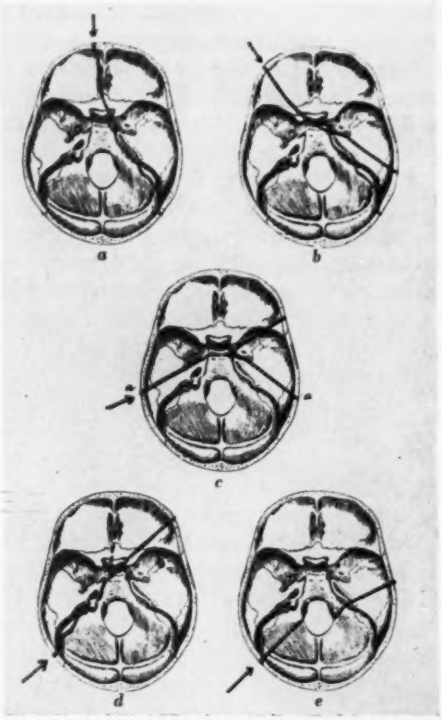

Fig. 1. Diagram of lines pursued by basic fractures. (a) Force applied to the median frontal region. (b) Force applied to the lateral frontal region in the situation of the external angular frontal process. (c) Force applied to the region of the external ear. (a - - - - a) The "typical" basic fracture. (d) Force applied to the mastoid region. (e) Force applied to the lateral occipital region.

of the mandible may be driven into the glenoid fossa, producing an extensive fracture of the floor of the middle fossa. The bony walls of the paranasal sinuses become thinner as age advances and pneumatization is completed; this, associated with the fact that bones become more brittle, makes

the base of the skull very vulnerable in the aged. A fatal blow may not produce any fractures but, on the other hand, extensive fractures may be present without any loss of consciousness.

MECHANICS OF SKULL FRACTURES.

Fractures of the skull in general are produced in accordance with certain well known laws. We know that the infant's skull is very elastic, but Bruns[6] was the first to call attention to the elastic properties of the adult skull, and Barnick[7] to the theory of von Wahl and Messerer, which is based upon these elastic properties. Von Wahl and Messerer divide all fractures caused by a blunt force into bending fractures (Biegungsbrüche) and bursting fractures (Berstungbrüche). A force applied to a circumscribed area of the skull will produce a bending type of fracture in its immediate neighborhood. The bursting or explosive type of fracture is caused by a broadly acting compressive force applied over a relatively large area of the skull and finds its greatest expression at the base. As Schönbauer and Brunner[8] point out, a broadly acting force produces compression of the skull in the direction of the acting force and spreads the skull at right angles to the direction of this force at the base, and here the bursting type of fracture is produced. Another factor which enters into the picture is the lack of uniformity in the structure of the base. Barnick,[7] Stenger,[9] and Schönbauer and Brunner[8] show that the base of the skull is its weakest portion, consisting of strong pillars or buttresses, such as the petrous pyramids and the wings of the sphenoid, with weaker intervening portions perforated by many foramina.

Besley,[10] Duret[11] and Poulain[12] do not accept the bursting theory on the ground that the skull is not a perfect sphere and, furthermore, has a fixed point where it articulates with the Atlas. According to them, when a blow is received anywhere on the vault there is corresponding counter-inbending force exerted at the articulation between the condyles and the Atlas. A careful study of 74 cases at autopsy convinced Besley[10] that fractures of the base are not produced by a bursting force but are due to a direct inbending force applied through the Atlocondylar articulation. How much more plausible this idea is when the force is communicated to the skull by a jar on the heels or a fall on the buttocks!

Ramadier and Caussé[13] speak of *a.* radiating fractures in which the petrous fracture is in continuity with one of the vault starting from the point where the force was applied, and *b.* independent petrous fractures where no fracture of the vault exists at or near the point of impact. The independent fractures were formerly considered to be very rare, but in 1921, Stewart[14] found that 30 per cent of the basal skull fractures were independent fractures, and in 1936 Voss[3] reported that 12 of 66 lateral fractures of the base were not combined with vault fractures. This type appears to be less rare than was formerly supposed, as one makes more use of the X-ray and of the microscope at autopsy. That indirect fracture is occasionally *contrecoup* in character is shown by a case reported by Ehrenfried[15] in 1923, in which there was a fracture of the left labyrinth from an injury to the right side of the head with no damage on the right side of the skull.

CLASSIFICATION OF SKULL FRACTURES.

The classification of skull fractures involving the ear presents a rather difficult problem, for we must take cognizance of the fact that in addition to actual fractures directly involving the temporal bone, the brain itself is also damaged by the injury and it is sometimes difficult to decide whether the damage to the ear function is due to actual fracture or to the concomitant brain injury, especially when this concerns the vestibular function of the ear.

In 1905, Passow[16] divided the pathological changes of ear injuries into: *1.* Fractures of the labyrinthine capsule; and *2.* hemorrhages into the labyrinth, or commotio labyrinthi.

In 1909, Stenger,[9] as the result of a study of his own cases, the study of pathological effects observed macroscopically and microscopically in experimental animals subjected to head trauma, and also a study of the literature, made the following classification: *1.* Damage to the inner ear with injury to the bony labyrinthine capsule; *2.* damage to the inner ear without any injury to the bony labyrinthine capsule; and *3.* damage to the inner ear without any demonstrable lesion of the skull. The first group comprises the transverse fractures of the pyramid. The second variety is caused by longitudinal fractures of the temporal bone. The third group of cases is called "Labyrintherschütterung" by Stenger.

In 1921, Ulrich[17] classified his cases as follows: *1.* Longitudinal fractures in which the capsule escapes, in which class the inner ear changes are characterized by hemorrhages in typical locations; *2.* transverse fractures, which as a rule are extensions of fractures of the posterior fossa or ring fractures of the base; and *3.* damage to the organ of hearing not caused by petrous fractures, in a few of which we have anatomical but no clinical findings. (One case of Ulrich's with rupture of the saccus endolymphaticus, cases of isolated cochlear fractures reported by Ulrich, Politzer, Manasse, de Kleyn and Stenvers and of Lothar Hoffmann.)

In 1925, Brunner,[18] feeling that not sufficient attention had been paid to the concomitant brain injury in previous classifications, divided his cases into: *1.* Commotio cerebri; *2.* Commotio cerebri et auris internae; and *3.* frakturae ossis petrosae.

The most recent classification of actual temporal bone fractures is that of Voss,[3] who divides these fractures into: *1.* Longitudinal fractures with injury to the middle ear; *2.* transverse fractures which involve the inner ear; *3.* combined oblique fractures of the posterior fossa and transverse fractures of the pyramid; *4.* combined oblique fractures of the posterior fossa and longitudinal fractures; *5.* combined longitudinal and transverse fractures of the petrous bone; *6.* partial or complete avulsion of the entire pyramid; *7.* partial or complete avulsion of the mastoid process; and *8.* isolated rupture of the tegmen tympani.

In 1934, Brunner[19] recognized these varieties of petrous fractures: *1.* Longitudinal fractures; *2.* transverse fractures; and *3.* fractures of the tip of the pyramid.

Inasmuch as my cases represent only clinical findings which are not supported by any pathological observations and have been studied solely from the standpoint of damage to the ear function, I shall use the following classification:

I. Fractures of the vault, base and face not involving the temporal bone in any way.

II. Longitudinal fractures of the petrous bone, unilateral and bilateral.

III. Transverse fractures of the petrous bone.

IV. Combined transverse and longitudinal fractures of the petrous bone.

I hope to be able to show that damage to the ear function, both cochlear and vestibular, can occur in Group I as well as in Groups II, III and IV.

PATHOLOGICAL ANATOMY.

Inasmuch as this paper represents a clinical study of skull fractures and is not supported by any pathological investigations, I must draw on the work of those who have made pathological studies of skull fracture cases, both recent and old, and of those who have conducted animal experimentation in the effort to learn just what takes place. The literature is very rich in this regard.

The otological symptomatology in skull fractures is difficult to understand unless one has a clear picture of the pathological changes produced by the trauma. In severe injuries which produce death, such knowledge can be obtained at the autopsy table; however, as Brunner[18] has pointed out, the pathological changes in patients who survive may be very different from those found in patients who succumb. For this purpose, animal experimentation must be resorted to. We are also fortunate in having autopsy reports of patients who have died months, even years, after the head injury from meningitis or some intercurrent condition.

In 1897, Barnick[7] made a histological study of the temporal bones of five cases of basal skull fracture. He states that most of the petrous fractures which come to autopsy are transverse fractures. He found numerous small hemorrhages in the acoustic and facial nerves, massive hemorrhages near the basal coil of the cochlea and in the region of the maculae acusticae, marked hemorrhages in the branches of the vestibular nerve and in the perilymph spaces of the semicircular canals. In all five cases he found hemorrhages in the narrow nerve canals leading to the otolith apparatus of the saccule and utricle and to the ampullae of the semicircular canals. Blood was rarely seen in the endolymph spaces. He calls attention to the fact that the round window is the only part of the labyrinth which can yield to increased intralabyrinthine pres-

sure and thinks that this is the probable explanation for the hemorrhages in its neighborhood.

Sakai[20] studied 10 temporal bones shortly after death from skull fractures and reported the following observations:

1. The bony labyrinthine capsule was not fractured in any case.

2. Hemorrhage occurred in the region of the round window membrane eight times, and at the oval window eight times.

3. The ligamentum annulare was torn only once.

4. The acustic nerve and its branches showed marked hemorrhage in all the cases. Of the two main trunks, the cochlear nerve showed a rupture of its fibres eight times, and the vestibular nerve four times.

5. There were hemorrhages into the soft parts of the utricle, saccule and canals five times.

6. Hemorrhage into the facial nerve occurred once.

7. Hemorrhage into the Fallopian canal was seen four times.

8. Hemorrhage into the ligamentum spirale occurred twice.

9. Hemorrhage into the ganglion spirale was seen twice.

In all of the cases of Barnick and Sakai, the changes in the soft parts of the labyrinth were very similar and consisted of large and small hemorrhages in certain sites of predilection, especially near the windows. All of the hemorrhages were into the perilymph. In reviewing his findings, Sakai expressed the opinion that similar changes, in patients who may recover, may later result in degenerative atrophy of the soft parts of the labyrinth and its nerve elements, or lead to the formation of new bone within the labyrinth, as was reported by Manasse and Nager (see below).

W. Lange[21] in presenting the microscopic findings of six cases of longitudinal fracture of the petrous bone, called attention to the following facts:

1. Infrequent damage to the bony labyrinthine capsule.

2. The tegmen tympani or antri was fractured in every case.

3. The tympanic membrane and external canal walls were often injured.

4. The oval and round windows were always intact.

5. There was always more or less blood in the depths of the porus acusticus internus.

6. Blood was found in the nerve canals, especially in the branches of the vestibular nerve.

7. Complete tearing of the acustic nerve was never seen, but separation of its fibres by blood extravasations was made out.

8. Damage to the facial nerve was not seen.

Ulrich,[22] in an exhaustive microscopic study of the temporal bones of 18 cases dying from basal skull fracture, found that the majority of temporal bone fractures coursed through the roof of the middle ear and then along the anterior edge of the pyramid. In all longitudinal fractures of the petrous bone, the labyrinthine capsule remained undamaged in every respect. The frequently damaged middle ear was always injured directly by the fracture line. If the facial canal was involved, the damage occurred in the region of the genu. In these longitudinal fractures the nerves to the inner ear were almost always injured by hemorrhage, tearing or stretching in the following frequency: vestibular, cochlear and, least of all, the facial. In contradistinction to the damage of the trunk of the vestibular nerve, the vestibular end-organ was rarely injured. There was, however, frequent evidence of injury to the interior of the cochlea. Hemorrhages into the ligamentum spirale and into the scala tympani immediately behind the round window were characteristic findings. Hemorrhages into the endolymph were never found.

According to Ulrich, the transverse fractures were always at right angles to the pyramid. The cochlea was more vulnerable than the vestibule. In contradistinction to the findings in longitudinal fractures, an actual tearing of the nerves at the internal auditory meatus may occur, as well as tearing of the membranous labyrinth, and also hemorrhages into the endolymph, as well as into the perilymph. The damage found in the middle ear affected only its mesial wall (canalis musculotubarius, knee of the facial, stapes footplate). The tearing and stretching of nerves is explained by Ulrich by the

fact that they are more or less fixed in their canals and the movement of the brain subjects them to traction, and he states "that in skull trauma in which cranial nerves are injured, their susceptibility to damage is inversely proportional to their relative length." For this reason the Nervus Ampularis is most frequently damaged by traction, and the Nervus Facialis least often.

PATHOLOGY IN EXPERIMENTAL ANIMALS.

As it may be argued that the pathological changes in those surviving their injuries may be different from those found in patients who succumb, a study of the pathological changes produced by head trauma in experimental animals becomes of vital importance.

Stenger[9] attempted to determine the changes which occur in the inner ear of animals after mild injuries. His experiments with white rats consisted of light blows of a hammer on the skull, and he tried not to fracture the skull. A variable degree of unconsciousness followed. After a variable period of time, sometimes as long as three to four weeks, the rats were killed by careful bleeding and the temporal bones were studied microscopically.

After light blows, hemorrhages were found in the region of the round windows and in the basal coil of the cochlea, whereas the vestibule showed no damage.

With more severe blows, the hemorrhages were more extensive and reached the tip of the cochlea, but were confined to the scala tympani. Bleeding was more marked at the round window; mild bleeding also occurred into the cochlear nerve, and occasionally into the ampullae.

With still more severe injuries, there were severe hemorrhages about the round windows, which seemed to be torn in some of the preparations, bleeding into the entire cochlea, marked bleeding between the fibres of the N. Acusticus, and hemorrhages in the ampullae. No direct damage to the temporal bones could be made out in any of these cases.

These experiments were repeated on guinea pigs by Brunner,[18] and his findings were quite similar to those of Stenger. He found no definite changes in the neuroepithelium of the inner ear but found definite pathology in the blood vessels

with hemorrhages in the perilymph and endolymph spaces, in the aqueducts and near the round window.

And so it becomes quite apparent that the pathology found in the experimental animals approximates very closely that found in patients dying shortly after injury, at least insofar as it concerns the vascular system. It seems rather fair to conclude that the same sort of pathology is present in patients who survive as in those who succumb to their injuries, although it may be of less severity in degree and it probably consists of hemorrhage and injury to nerves. One could only conjecture the end-results of these lesions upon the organ of hearing were it not for the published cases of Theodore and Manasse, which give us the clinical and pathological picture of individuals who died at more or less remote periods after head trauma.

Case of Theodore:[23] R. E., age 42 years, was struck by a bicycle on Nov. 12, 1896, his head striking the pavement. He was unconscious for 12 to 14 hours. On awakening, he had severe headaches, vertigo and vomiting whenever he raised his head. There was bleeding from the right ear. It was ascertained that in 1886 he had received another blow to the head, following which his hearing was not so good as it had been.

Examination: In the right occiput there was a contused wound with edematous edges. The right mastoid was tender. Fissures could not be felt. There was no vomiting. Marked headache was present. Vertigo was still marked in the recumbent as well as sitting or standing posture. The Romberg sign was negative. On forward gait with eyes closed, there was considerable staggering to both sides. There was suggilation behind the right ear to the tip of the mastoid. Tests of hearing gave the following results:

TABLE I.

Test of Hearing	Right Ear	Left Ear
Whisper	Not heard	0.75 meter
C fork	Not heard	Heard
C_1 fork	Hearing weak	Heard
Galton whistle	Normal	Heard
Weber test	Referred to right ear	
Bone conduction	4 seconds short	Normal

Eye signs were not present.

By Dec. 5, 1896, the swelling of the posterior canal wall of the right ear had receded. The drum was visible and hemorrhagic. Whisper was heard at 10 cm. in the right ear.

By Jan. 7, 1897, headaches only recurred after exertion and excitement. Vertigo entirely gone. Right external canal normal. Right drum dull and thickened. Hearing for whisper, 25 cm. on right, and 0.75 m. on left. C_1 fork heard in both ears. Weber not lateralized. Rinné positive on the right.

In 1908, patient was readmitted to the hospital because of pulmonary tuberculosis. At this time examination showed both drums thickened and retracted. Results of the hearing tests were as follows: Whisper not heard with either ear. Conversations heard by each ear at the ear; all forks heard; Galton whistle, right 2.0, and left, 1.8; Weber not lateralized; Schwabach, right 10 seconds short; left, seven seconds short.

Death occurred on Feb. 27, 1908.

Section (Prof. Chiari): No signs of skull fracture or fractures of the temporal bones could be made out.

Microscopic Examination: The right petrous bone showed an intact labyrinthine capsule. Everything was found to be normal except the cochlea, and especially the basal coil. Corti's organ in the basal coil consisted of merely a heap of cells in which details could not be distinguished. The inner hair cells were replaced by round and cubical cells with well defined nuclei, the cytoplasm being partly hyaline or granular. Similar changes were also found in the outer hair cells. In some sections the membrana tectoria lay tight against Corti's organ, resulting in a marked reduction of the lumen of the ductus cochlearis. The ligamentum spirale was poor in cells. The most marked change was in the spiral ganglion of the basal coil. Its few remaining cells were shrunken, with little protoplasm and small or no nuclei. In the nerve canals there was a marked thickening of the inner periosteum due to connective tissue showing hyaline degeneration. The nerve bundles lying in this hyaline sheath were atrophic.

The observations on the left temporal bone were identical with those in the right.

This is the picture of a man who had sustained two injuries to his head, 12 and 22 years before his death from an inter-

current disease. On the occasion of his second injury, he must have suffered a right longitudinal fracture of the petrous bone with massive bleeding into the right inner ear. After the usual improvement in hearing to be expected in the first few weeks after the trauma, the hearing gradually diminished, and at section marked atrophic degenerative changes were found in the neuroepithelium of Corti's organ and the spiral ganglion, particularly in the basal coil. The end-arborization of the cochlear nerve was degenerated and attenuated, and the fine bony canals housing these nerves were partly obliterated by a hyaline connective tissue. No evidence of a fracture could be made out in the right temporal bone, bearing out an observation made by Ulrich that petrous bones show more tendency to heal by bony union the more one approaches the periphery of the bone. These general observations of Theodore were, therefore, similar to those made by Alexander[24] 44 days following a head injury, only more extensive, and the pathology found by Theodore was also similar to the changes found by Manasse in all of his cases of progressive labyrinthine deafness.

Equally illuminating is a case report of Manasse.[25]

Case Report: A man, age 39 years, had been injured 15 years before death by a fall on the head, resulting in complete deafness. Death resulted from pneumonia. The observations were as follows:

1. Nothing pathological was found in the skull or the dura.

2. Fine fissures in the labyrinthine region of both petrous bones were seen microscopically. No fissures were visible macroscopically. Those observed affected the external wall of the labyrinth on both sides, running through round and oval windows. They were not united by bone or callus but were filled with connective tissue. The cochlea was spared by the fissures.

3. A bony new growth was seen inside of the labyrinth which was symmetrical on both sides. This process was the same in the cochlea as in the vestibule, although the vestibule was involved more than the cochlea, which fact may be attributed to the localization of the fissures. The membranous semicircular canals were entirely obliterated by the new growth. Manasse calls this process a periostitis obliterans.

4. The fourth significant observation was the atrophic degeneration of the nervous elements, the ductus cochlearis being most affected. There was high grade degeneration of the organ of Corti and of the cochlear nerve and its branches. Similar degenerative changes were seen in the spiral ganglion.

This case was one of bilateral labyrinthine fracture in which the fissures were so fine that they escaped macroscopic observation, and yet the changes in the nerve and neuroepithelium were similar to those observed by Theodore in his case of longitudinal fracture. That such microscopic fissures often exist was also stressed by Schönbauer and Brunner,[8] Ulrich,[22] Voss[3] and others. A second significant observation was that even after 15 years the fissures were not united by a bony union.

Taking into consideration the pathological changes found in those dying soon after their injuries, the changes found microscopically in the experimental animals, and the changes observed in the temporal bones of those who die many years after their trauma the pathology in all injuries to the temporal bone must be very similar and consist of stretching and tearing of nerves to various degrees, hemorrhages in various locations, the site of predilection being the perilymph spaces of the basal coil of the cochlea in the vicinity of the round window. The exudation of blood is probably partly absorbed and partly organized, leading to atrophy of the finer nerve branches and degeneration of the neuroepithelium of both cochlea and vestibule.

Combined fractures of the vault and the base are said to be much more numerous than isolated fractures of the base. In a series of 104 cases of middle fossa or petrous fractures, Voss[3] found that 79.2 per cent were combined vault and base fractures, whereas only 20.8 per cent were pure, isolated basal skull fractures. This opinion is supported by Schönbauer and Brunner,[8] who found, in a study of 57 macerated skulls, that four-fifths of the fractures were combined vault and base fractures. Usually the extension of the basal fracture into the vault or of the vault fracture into the base was so fine that it could only be seen in the macerated skull.

The majority of basal skull fractures occur in the middle fossa. Thus, Schönbauer and Brunner[8] found, in their study

of 57 macerated skulls of basal skull fractures, that 45 of them, or 80 per cent, were middle fossa fractures, as against 60 per cent which had been reported by Matti.

The classical varieties of petrous bone fractures are longitudinal, transverse and combined longitudinal and transverse; of these, the longitudinal variety is by far the most numerous. Biechele,[20] reporting the material of the Würtzburg Clinic, found longitudinal fracture present in 55 per cent of 74 cases. Schönbauer and Brunner[8] reported 14 cases of longitudinal and three of transverse fractures among 23 cases, and Voss[3] reporting upon 63 cases of petrous fractures, found the longitudinal variety present 46 times, the transverse six times, and the combined longitudinal and transverse six times. In my own series the longitudinal fractures were even more predominant, there being 146 cases of longitudinal, eight cases of transverse, and eight of combined longitudinal and transverse fractures.

THE PATHOLOGY OF LONGITUDINAL FRACTURES.

According to most authors, the course of the longitudinal fracture is quite typical. This fracture, parallel to the long axis of the pyramid (Ramadier and Caussé[13]) is usually due to a force applied to the temporoparietal region; descending from this region, the fracture passes in front of or at the level of the external auditory canal and ends at the anterior lacerated foramen or the region of the Gasserian ganglion. It originates in the squama or in the parietal region behind the squama; in the first instance it passes through the roof of the tympanum and at the same time through the roof of the external auditory canal; when it originates in the parietal bone it affects first the superior part of the mastoid and then passes through the superior and posterior walls of the canal and through the superior wall of the tympanum. According to Voss,[3] it may bifurcate into two branches, limiting a raised or broken fragment of bone; one branch directs itself toward the temporomandibular articulation, the other runs inward and forward toward the tegmen tympani, whence it courses along the anterior aspect of the pyramid to the fossa of the Gasserian ganglion, passing through the superior wall of the Eustachian tube and the carotid canal. The middle ear is always damaged. The drum is torn, and bleeding occurs.

The thin roof of the tensor tympani muscle and the Eustachian tube lie in the line of this fracture. The incus may be dislocated and the ossicular ligaments torn. The labyrinth capsule remains undamaged, although marked perilymphatic hemorrhage in the scala tympani may take place, particularly near the round window. The facial nerve is usually spared, although, as Brunner and Ulrich point out, the geniculate ganglion may be in the pathway of the fracture. Fig. 2, reproduced from Voss,[3] represents a pathological specimen obtained at autopsy in an individual who did not die from his skull fracture. It shows an old, unhealed longitudinal fracture

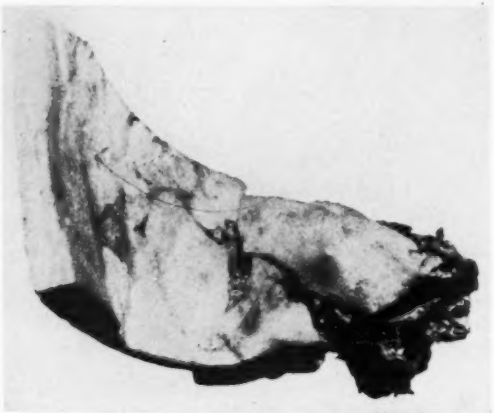

Fig. 2. Longitudinal temporal bone fracture. (Otto Voss: Die Chirurgie der Schadelbasisfrakturen. Leipzig.)

coursing through the tegmen tympani and the roof of the Eustachian tube.

Exceptionally, the fracture may extend to the opposite side. After passing through the sphenobasilar suture, it mounts up along the anterior aspect of the other pyramid, a bilateral longitudinal fracture. In my own series of 146 cases of longitudinal fractures, 34 were bilateral. Usually the fracture line is large and easily discernible macroscopically, but at times the fissures are extremely fine and can be discovered only by the magnifying glass, microscopically, or in the macerated skull (Schönbauer and Brunner,[8] Guerinot,[27] Voss[3]).

THE PATHOLOGY OF TRANSVERSE FRACTURES.

While longitudinal fractures of the petrous bone usually pursue a rather typical course, the same cannot be said of the transverse variety. These fractures, generally the result of force applied to the occipital or occipitomastoid regions, are for the most part posterior fossa fractures. They usually begin in the posterior fossa and, as the name implies, cut across the pyramid transversely, but not always at right angle to the axis of the pyramid as Ulrich[22] contends. According to Proust[28] and Félizet,[29] they run from the external part of the posterior lacerated foramen (jugular) to the anterior lacerated foramen or the small round foramen (see Fig. 3).

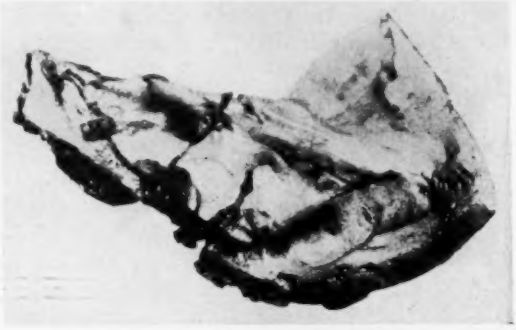

Fig. 3. Transverse fracture through the labyrinth. (Otto Voss: Die Chirurgie der Schadelbasisfrakturen. Leipzig.)

As a matter of fact, transverse fractures are the subject of considerable variation in position and direction. There are two extreme types: first, the internal, which traverses the internal auditory meatus and at the same time shatters the cochlea in its anterointernal portion; and second, the external type, which passes through the entire internal ear, cochlea and vestibule, and through the Fallopian canal. The middle ear may be entirely spared but if it is injured at all the damage affects only its medial wall (the Eustachian tube, the knee of the facial and the footplate of the stapes — Ulrich[22]). Fissures are frequently found between the windows; the promontory may be torn loose; the footplate of the stapes may be luxated. In two of the four cases operated upon by Voss,[3] the

lateral wall of the internal ear was involved. They may cause a hematotympanum by injury of the mesial wall of the middle ear, but they rarely, if ever, cause a rupture of the drum and bleeding from the external canal. As the fracture courses through the labyrinthine capsule, it usually causes complete loss of function, both cochlear and vestibular, by direct injury to the membranous soft parts, by hemorrhage into the hollow spaces, and by tearing or sectioning the acustic nerve. The facial nerve is especially vulnerable, either at the internal auditory meatus or in the second portion of the Fallopian canal. The transverse fracture is essentially a fracture of the labyrinth.

The labyrinthine fracture which puts the inner ear into communication with the middle ear and the Eustachian tube is especially dangerous from the standpoint of post-traumatic meningitis, a subject which will be discussed further under the head of prognosis.

THE PATHOLOGY OF COMBINED TRANSVERSE AND LONGITUDINAL FRACTURES.

This type, also called oblique by Ramadier, combines the features of both varieties. That these combined fractures are not rare is shown by the reports of Voss[3] and Ulrich.[30] The statistics of Voss give six combined fractures in 63 cases of temporal bone fractures, and Ulrich reported two among eight cases of labyrinthine fracture. Biechele[26] reported 14 cases of combined longitudinal and transverse fractures in 74 cases of temporal bone fractures seen during a period of four years at the clinic of the University of Würtzburg. The transverse fracture was combined with the longitudinal variety in 16 of my cases.

THE PATHOLOGY OF MICROSCOPIC FRACTURES OF THE LABYRINTHINE CAPSULE.

Microscopic labyrinthine fractures have been carefully studied and reported by Schönbauer and Brunner,[8] Nager,[31] Ulrich[30] and Voss.[3] They are invisible at autopsy, though for the most part visible in the Roentgenogram and always histologically. This fracture may take the typical course of the transverse fracture but it may also remain strictly intracap-

sular. Among eight cases of Ulrich's which were visible by X-ray, five were transverse and three purely intracapsular. Halphen[32] designates the subvestibular portion of the tympanic slope as the most feeble portion of the labyrinthine shell. Capsular fractures have their seat of predilection in the region of the windows. They break the osseous bridge between them, shatter the stapes, and tear the round window. They may have several lines, as preparations of Nager and Ulrich show. A case of contralateral isolated capsular fracture has been reported by Ehrenfried,[15] and a bilateral capsular fracture by Voss.[3]

THE PATHOLOGY OF ISOLATED LABYRINTHINE FRACTURES.

Until a fairly recent date it was believed that if the labyrinthine shell was fractured at any point, a complete cochleovestibular paralysis would ensue. While this is true in the vast majority of cases, it must now be conceded that it is not true in every case, and that isolated fracture of either cochlea or vestibule may occur with an isolated loss of function of one portion and a retention, or at least partial retention, of function of the other portion of the labyrinth.

Uffenorde[33] (cited by Ramadier) was able to show, by anatomical and pathological studies, that hemorrhage into the soft parts of the labyrinth, produced by a line of fracture, can absorb with a return of at least partial function. These researches would indicate that a labyrinthine fracture can result in only a partial paralysis of that organ. In 1929, Klingenberg,[34] of Nager's Clinic, called attention to the cases reported by Politzer,[35] Manasse and Ulrich, in which an isolated cochlear fracture was found pathologically, but in which nothing was known about the clinical findings. He further called attention to cases reported by De Kleyn and Stenvers[36] and Lothar Hoffmann,[37] in which the clinical picture, including X-ray findings, was that of an isolated cochlear fracture, but in these cases there were no pathological examinations. Furthermore, the case of De Kleyn and Stenvers is not free of criticism because an otosclerosis existed before the injury and may of itself have been responsible for the total loss of hearing in the right ear, in which the fracture was indicated by X-ray. And in the case of Lothar Hoffmann, although X-ray examination revealed a fracture of the right pyramid, and the

right ear was totally deaf, the state of the vestibular function was unknown.

Klingenberg[34] himself reported two cases of his own in which there was a complete unilateral deafness with at least a partial retention of caloric irritability. He believes that his cases indicate that the cochlea is more vulnerable to fracture than the vestibule and that the retention of some vestibular function in these cases can be explained by a closure of the ductus reuniens, due to reaction.

Much more illuminating and conclusive than any of these cases is one reported by Schittler,[38] of an individual dying of meningitis, who had received an injury to his head 16 years before, following which he had been unconscious for five minutes and had bled from the right ear. Repeated examinations in the interim had shown a total loss of caloric irritability with retention of somewhat reduced hearing ability. Microscopic examination of the temporal bone revealed two fractures involving the vestibule but sparing the cochlea. The opportunity for watching such a case through the initial injury and then making a pathological examination of the temporal bone many years later does not often arise, but this case proves rather conclusively that an isolated fracture of the labyrinth capsule can occur and that, as a result, one of the labyrinthine functions can be retained. It further proves that the opinion of Ulrich,[22] expressed in 1926, that the anatomical basis for the isolated loss of function of the cochlea or the vestibule lies in a separate injury to one or the other branch of the acustic nerve and not in a partial labyrinthine fracture, is not entirely correct. In 1933, Biechele[26] reported that in three of the 27 cases of labyrinthine fracture observed at the Würtzburg Clinic, the vestibular function was present. Lange is also of the opinion that labyrinthine fractures can involve single parts of the labyrinth.

In three of the 16 cases of fracture of the labyrinthine capsule in my own series, I have reason to feel that we are dealing with an isolated cochlear fracture, although I feel that this diagnosis cannot be made with absolute certainty in any case unless the temporal bone can be examined microscopically. I shall report these three cases briefly:

Case 99: Henry S., age 53 years, was injured, March 11, 1938. He fell down some steps and was unconscious for about six hours. He bled from his left ear.

Examination on June 17, 1938: He complained of total loss of hearing in the left, and defective hearing in the right ear. He also complained of attacks of vertigo. There was a spontaneous rotary-horizontal nystagmus to the right. There was

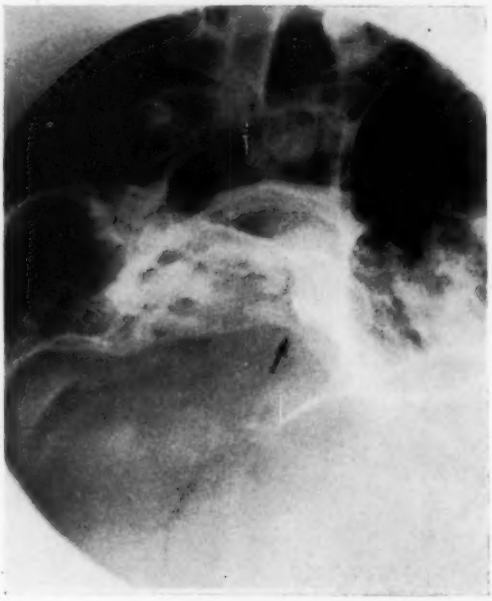

Fig. 4. X-ray (Stenver's position) showing isolated cochlear fracture of Henry S., Case 99.

total deafness of the left ear to whisper, voice and audiometer with the right ear properly excluded. The right ear showed a marked combined type of deafness. Caloric tests: 5 cc. of water at 80° F. in the right ear produced a definite nystagmus in 10 seconds; 5 cc. of water at 80° F. in the left ear produced only a few nystagmoid jerks, but 10 cc. produced a typical response in 58 seconds. The X-ray (Epperson) revealed "no fracture of the vault in stereoscopic lateral views but in Sten-

vers' position did show an oblique line passing upward and backward through the left petrous bone from a point on the lower border 20 mm. from the apex, and emerging at the upper border about 25 mm. from the apex."

Case 211: Louis L., age 47 years, was injured on Aug. 15, 1937. The right side of his head was struck by a large piece of steel being cut by shears. A short period of unconsciousness followed. There was no bleeding from nose, mouth or ears, but he expectorated some blood.

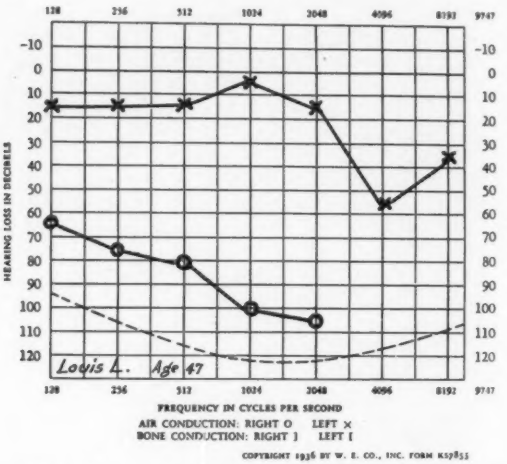

Fig. 5. Audiogram of Louis L., Case 211, showing some remnant of hearing in the right ear.

Examination on March 16, 1938: There was no facial paralysis. Whisper heard one foot with right ear, and 25 feet with left. Audiometer showed a subtotal loss of hearing in the right ear and a moderate perception loss in the left. Caloric tests gave no response in the right ear, and a normal response in 15 seconds in the left ear with the same stimulus.

Examination on March 26, 1938, showed a total deafness for the right ear (Barany apparatus in left) on the audiometer with a moderate perception loss on the left; 20 cc. of water at 64° in the right ear produced a typical nystagmus but no vertigo.

Examination on Sept. 21, 1938, showed the same total deafness in the right ear as was present on March 16, proper exclusion being used for both voice and audiometer tests, and water at 56° F. produced both nystagmus and vertigo in 33 seconds. X-ray examination in Stenver's position (Epperson) revealed an irregular oblique line in the right petrous bone and also a fissure running from the base into the squama of the right temporal bone.

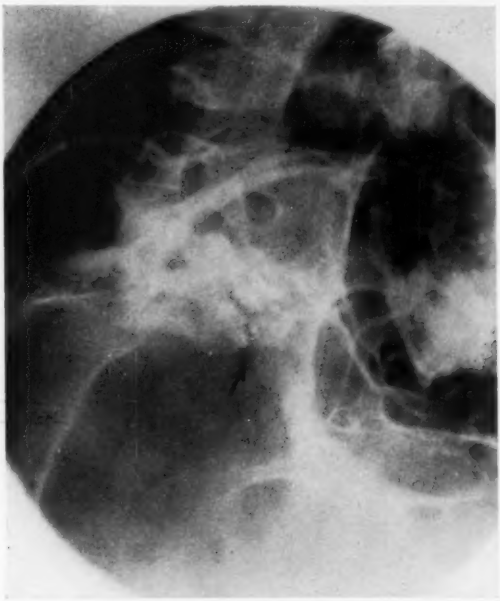

Fig. 6. X-ray (Stenver's position) showing isolated cochlear fracture of Louis L., Case 211.

Case 118: Fred W., age 53 years, was injured on Oct. 18, 1937, when he fell a distance of 35-40 feet while cleaning windows. There was a period of unconsciousness. When he recovered consciousness he was bleeding from the right ear, and he vomited blood several times. X-rays taken at the Emergency Hospital and later at another hospital were negative for skull fracture.

Examination on Jan. 13, 1938: No facial paralysis present. Hearing examination showed right ear totally deaf to voice and the audiometer, with fairly normal hearing on the left. There were present spontaneous nystagmus, pastpointing, drift in the deviation test, and a suggestive Romberg. Both labyrinths reacted to stimulation by 5 cc. of water at 80° F. On March 2, 1938, the hearing was again tested, with the same results.

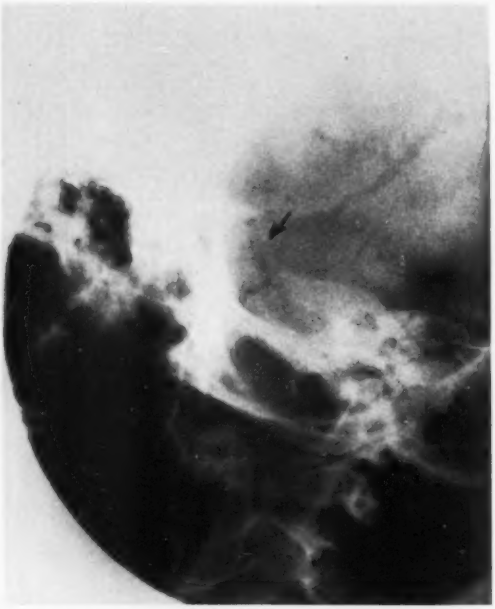

Fig. 7. X-ray (Stenver's position) showing isolated cochlear fracture of Fred W., Case 118.

Examination on May 23, 1938: The hearing was again rechecked and with the same result as on previous tests. In the caloric test, the right ear did not react to 5 cc. of water at 80° F., but did react to 20 cc. of water at 40° F. The X-ray examination in Stenver's position showed an irregular transverse fracture of the right petrous bone, 11 mm. from the apex.

Comment: These three cases are probably isolated cochlear fractures. The absolute diagnosis of an isolated cochlear fracture can only be made, in my opinion, if such clinical facts as I have reported are supported by a pathological examination which includes a microscopic study of the temporal bone. In all three there was unilateral total deafness with retention of the vestibular function, although it was subnormal. Case 211 is interesting, in that the ablation of cochlear function was not sudden, but gradual. Seven months after injury, he was still able to hear a whisper at one foot in the affected ear. This case is an example of a pure capsular fracture with isolated loss of cochlear function, but in Cases 99 and 118 the isolated labyrinthine fracture was combined with a longitudinal fracture of the temporal bone.

MASTOID FRACTURES.

Ramadier and Caussé[13] call attention to a type of temporal bone fracture which I myself have not observed. This fracture is restricted to the mastoid itself and is quite rare. It can open into the external auditory canal and the middle ear. It can also reach the facial nerve in its descending portion. In two cases reported by Boullet, the apophysis was detached from the rest of the temporal bone and pulled downward by the action of the sternomastoid muscle. Passow,[16] Walb and Mauthner[39] are responsible for the observation that when the skull is compressed tranversely, a bilateral mastoid fracture due to crushing may result, and note that this type of fracture is not rare among miners. Mauthner reviewed 45 such cases.

END-RESULTS OF HEMORRHAGE INTO THE INNER EAR.

From the pathological studies of various investigators (Ulrich,[22] Nager,[31] Schönbauer and Brunner,[8] Brunner,[18] Brock,[40] Voss,[3] W. Lange,[21] Barnick,[7] Sakai[20] and others) we know that temporal bone fractures, both longitudinal and labyrinthine, cause an effusion of blood into the hollow spaces of the internal ear, and from the experimental studies of Stenger,[9] Brunner[18] and Yoshii,[41] we learn that this can occur even without fracture. What happens to this effusion of blood and what result does it have on the functions of the ear? Manasse[25] answers this question for us in reporting the path-

ological findings in the temporal bones of two cases, one of Kundradt[42] and one of his own, in which two head traumas had been suffered 10 and 15 years, respectively, before death. In Kundradt's case the labyrinth showed a complete bony obliteration. In his own patient, dead from a pneumonia, the skull was macroscopically negative for fracture. Microscopically, however, there were fine fissures on the lateral labyrinthine walls of both petrous bones. They ran through the round and oval windows of the vestibule. Even after 15 years they were easily visible microscopically and were not united by bone formation. The cochlea was entirely spared by the fissures. There was a bony new growth inside of the labyrinth, symmetrical on both sides and affecting the vestibule to a greater extent than the cochlea. This consisted of three layers: *1.* A connective tissue layer; *2.* a layer of new bone; and *3.* a layer of older bone tissue. Of these, the last was nearer the periosteum of the canals. The membranous semicircular canals were almost completely obliterated by the osseous new growth. He called the condition a periostitis obliterans, the same condition to which Nager gives the name otitis chronica obliterans.

Another significant feature in the case was the atrophic degeneration of the nervous elements. The ductus cochlearis suffered the most, showing a high grade degeneration of Corti's organ. The cochlear nerve and its branches, as well as the cells of the spiral ganglion, showed similar changes. According to Manasse, similar changes are found in cases of chronic progressive deafness and in deaf-mutism. Thus, we see that while small hemorrhages into the inner ear can probably be absorbed, as Uffenorde has pointed out, more extensive hemorrhage leads to chronic degenerative changes which profoundly affect the function of the ear.

THE REPAIR OF PETROUS FRACTURES.

The reparative processes in fractures of the base and, more particularly, in fractures of the temporal bone are different than they are in other bones of the body or of the vault of the skull. This fact was commented upon by Bruns[6] as early as 1886. The closure of the fracture by bony callus is either incomplete or fails entirely. This repair of the fissure by fibrous tissue instead of by bony callus seems to apply

to both longitudinal and transverse fractures of the petrous bone, although the tendency to fibrous union appears to be more marked in the transverse variety. In 1933, Lindemann,[79] of Voss' Clinic, made a complete survey of the literature covering this question, and the consensus of opinion was that basal skull fractures, in general, and petrous fractures, in particular, show little tendency to bone consolidation, although some authors have described cases in which fractures in the neighborhood of the pyramid had completely healed by bony union (Frobes, Stenger, Kundradt, Richet, Chesten, Morris, Katz, Runge, Scarpa and Kuster).[79] Lindemann X-rayed 30 cases of all sorts of skull fractures at periods of one to 25 years after injury and found that the fractures were recognizable by X-ray in nearly all of them.

Speaking of longitudinal fractures, Brunner[19] found in 12 days after the accident a closure consisting of young connective tissue. Ramadier and Caussé[13] examined a longitudinal fracture 15 months after injury and found it closed by dense, firm, fibrous tissue, strongly adherent to the osseous edges. Alexander[24] found only a fibrous closure in a case 31 years after injury. From these and other findings, we must conclude that longitudinal fractures, in the main, do not close by a complete bony callus.

The tendency to fibrous union is even more marked in transverse fractures. Manasse,[25] Ulrich,[22] Schönbauer and Brunner[8] and Nager[44] have clarified the question of the consolidation of petrous (labyrinthine) fractures by microscopic examinations on temporal bones removed at autopsy. They showed that the defect in bone consolidation did not equally affect the entire extent of the fissure. In the peripheral portion, near the petrous cortex, the fissures were completely closed by bony callus. On the contrary, in the central portion, *i.e.*, at the level of the tympanic wall of the internal ear (promontory and region of the windows), it was closed solely by connective tissue. In the region of the footplate of the stapes the interfragmentary part often remained wide open, even fibrous repair being scarcely half finished. Their histological sections prove indisputably that such a defect can permit the passage of an infection from the middle to the inner ear.

The researches of Nager[44] have given the histological and biological characteristics of the bony labyrinthine capsule

which explain this interesting phenomenon of the nonconsolidation of capsular fracture lines. According to Nager's studies the labyrinthine capsule is composed of three distinct layers: *a.* an endosteal layer (internal) clothing the labyrinthine cavities and formed of embryonic bone; *b.* a periosteal layer (external) formed of diploic bone, like the rest of the skull, and capable, in case of fracture, of repairing itself by bony union. This layer is very thick toward the periphery in the neighborhood of the dura but at certain points in the region of the promontory and the windows, it is very thin and may even be dehiscent. *c.* The enchondral, or middle layer, is the thickest of the three. Although the process of enchondral ossification ends in the production of adult lamellar tissue in the rest of the skeleton, this is not true in the labyrinthine capsule. According to Nager, the enchondral embryonic bone remains throughout life as primary bone and in case of fracture it exhibits no power of osseous regeneration.

Prof. Bast,[78] of the University of Wisconsin, who has done considerable work on the development of the otic capsule, is responsible for the following statements: "The enchondral layer of bone does not change much after birth, except that it becomes somewhat denser. It is not uniformly distributed throughout the otic capsule but is fairly abundant in the region of the round window, anterior and posterior to the oval window, in the deeper part around the internal auditory meatus, and around the first and second turns of the cochlea. It is fairly abundant around some of the semicircular canals. There are marked differences in the thickness of the otic capsule in different individuals. This applies both to the enchondral bone as well as to the periosteal and endosteal bone . . . already early in the development of the capsule one can note these differences. . . . In the regions in which enchondral bone predominates, there is usually a paucity of periosteal bone. I do not know why enchondral bone has so little ability to regenerate in the otic capsule; however, this does not apply only to enchondral bone but periosteal bone likewise has poor regenerating powers in the region of the otic capsule. Both of these bones, and especially the enchondral bone, have a scant blood supply. The bone is very compact and vascular channels are far apart. This extreme compression may have caused the dormant osteoblasts to have lost

their vitality. There is a semblance of Haversian systems in enchondral bone but it is of the primary bone type."

These facts relating to the development and the adult form of the otic capsule furnish the biological key to the repair of fractures in this region. What bearing this peculiar characteristic has on the question of the prognosis of labyrinthine fractures will be more fully discussed below.

SYMPTOMATOLOGY AND DIAGNOSIS.

For the diagnosis of ear fractures, longitudinal or transverse, partial or complete, a careful evaluation of the symptoms and signs is necessary. These consist of an early otoscopic examination by an otologist, history of unconsciousness, presence of the discharge of blood or cerebrospinal fluid from the ear, presence of hematotympanum or liquor tympanum, lumbar puncture, presence of ecchymosis over the mastoid, facial paralysis or paresis, functional testing and a radiological examination.

UNCONSCIOUSNESS.

There usually is a variable period of unconsciousness following an injury to the head of sufficient intensity to produce a fracture of the skull. But, as Mellenger[4] has pointed out, this is not absolutely necessary even in rather severe fractures of the skull. Ordinarily, however, the longer the period of unconsciousness the more serious the injury. In 28 cases of my series of 211 skull fractures, there was no period of loss of consciousness, and in nine further cases the history on this point was indefinite.

(To be concluded in September, 1939 issue.)

A THYRATRON INFLECTOR, ITS BEHAVIOR WITH CERTAIN VOWELS AND ITS USE IN INSTRUCTING DEAF CHILDREN.*†

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I.–INTRODUCTION.

It is an important aim of the teacher of deaf children to make their speech as intelligible and as pleasing to the ear as possible. A deaf individual may master perfectly the mechanics of the individual speech elements; yet lack of normal emphasis and inflection will make his speech difficult to understand. Furthermore, it frequently happens that a deaf child's voice is badly pitched, the speech then being distressing to those who hear him. The deaf person has to rely mainly on kinesthetic and touch sensations for perception of pitch. He may feel differences in pitch in vibrating bodies, such as the sounding board of a piano, or in apparatus specially constructed for his instruction, such as the Gault teletactor;[1] and perception of pitch difference in his own voice comes from the kinesthetic sensations in the vocal musculature.

The underlying principle in educating the deaf is to substitute where possible the use of other senses (in practice, touch and vision) for perception of such stimuli as are normally perceived through the ear. Lip-reading (or speech-reading) can to some extent supplant the normal method of hearing the conversation of others. In forming speech sounds, however, hearing persons are guided by the fact that they hear themselves and the speech of those around them; whereas a deaf child learns speech mainly through the process of associating the kinesthetic sensations derived from making certain sounds with the approval of the teacher when the correct movements and combinations of movements are made. Lip-reading helps the deaf person to understand others, but he has little to guide

*Candidate's Thesis presented for the Degree of Master of Science in Education, Washington University, St. Louis.

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Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 11, 1939.

him when he himself attempts to speak. Many educators of the deaf have, therefore, been concerned with attempts to provide their pupils with some means of enabling them to see positive and definite effects created by the sounds they produce. The apparatus described in this paper is one in which a vertical row of lights is arranged to flash on in accordance with the fundamental pitch of the voice — the higher the pitch, the greater the number of lights which respond. The effect is that of a column of light whose height indicates the pitch level. Voice and the inflection of the voice are thus translated into visual symbols.

II.–HISTORY.

The first teacher of the deaf to realize the educational possibilities of instruments which translate sound into light impulses seems to have been Alexander Graham Bell. In 1873,[2] he became interested in the "manometric capsule" of Koenig.

"It consisted of a cavity in a piece of wood, divided into two portions by a partition, or diaphragm, of gold-beater's skin. To one compartment was connected a gas pipe, so that it could be filled with gas, which was lighted at a burner let into one side of the capsule. The other compartment was connected with a speaking tube. Whenever a noise was made in the tube, the vibrations of the air were communicated, through the membrane, to the gas and thence to the flame. When vocal sounds were uttered in the tube, the flame moved up and down just as many hundred times per second as the voice vibrated."

Bell was favorably impressed by this apparatus and considered that it might be useful in teaching a deaf child correct articulation.

Scripture[3] reported on the use of the manometric capsule in correcting the speech of the deaf:

"The tone of the voice, or the laryngeal tone, is always imperfect in the deaf, even after the best instruction. It is too weak or too loud, too husky or too breathy or too tight, too high or too low, etc. It is always monotonous and devoid of melody."

Scripture gives a description of Koenig's manometric flame, and states that the breadth of the vibrations as seen in the revolving mirror depends on the "tone" at which the sound is made. His apparatus, the "organ trainer," comprised two "flame boxes" placed close together.[4]

"One of these is connected to an organ in such a way that the vibrations of the reeds are communicated to the flame. The person sings in the other one. When the mirror is turned, two bands of vibration are seen. It is easy to make the deaf child understand that his flame vibrations must match the organ vibrations. When middle C is played, his vibrations must be wide. He naturally tries all sorts of tones, but is soon able to make a tone with wide vibrations — that is, he sings middle C. With upper C, he tries till he matches the finer vibrations."

Scripture speaks in enthusiastic terms of the educational value of this instrument, and reports that it was used successfully with a large number of deaf pupils.

The strobilion, devised by Scripture in 1912, is also described in this book.[5] It consists essentially of a disc marked in concentric rings of alternating black and white spaces. The number of spaces in the rings increases from centre to circumference. The disc is made to revolve by means of an electric motor, and is illuminated by the flame from a "flame box." When a tone is sung into the mouthpiece of the flame box, the period of the flicker will synchronize with the period of the alternating spaces in one of the rings, which will, therefore, appear stationary. The position of the apparently stationary ring will then be an indication of the frequency of the tone. The principle of the strobilion is based on the "phonoskop" of Forchhammer,[6] a Danish physicist, who produced it in 1887.

Meyer[7] made certain modifications in the strobilion in order to increase its practicability as a teaching instrument:

1. The long rubber hose with its inflated rubber mouthpiece, of Scripture's design, was replaced by a short aluminum funnel, into which could fit the speaker's chin, mouth and nose. Nasal sounds were thus not excluded.

2. Scripture's disc, in which the number of spaces in the rings corresponded with the frequencies of the diatonic scale,

was replaced by one in which the number of spaces in each ring was one more than in the preceding ring.

3. In Scripture's apparatus the whole disc was visible. Meyer screened off all but a vertical strip, so that pitch level was indicated by the position of the stationary row of spaces on the strip — the conception of "high" and "low" voice could then be more readily grasped by a child.

4. An attempt was made to prevent the confusion caused by the instrument's response to the overtones in the voice. This effect was to some extent overcome by reducing the length of the vertical strip to two and one-half inches.

Meyer[8] gave records of his use of the strobilion with two children. He found it of considerable value, both in encouraging vocalization and in giving a conception of pitch and inflection.

Ferreri[9] described the "audiphone stroboscope," a multiple instrument used at the School for Poor Deaf Children in Milan. The output from a microphone was amplified, and part of the amplified sound was fed to a lamp which flickered synchronously. The lamp illuminated a stroboscopic disc, on which were painted differently colored rings. This instrument was used to indicate—

"the fundamental notes and harmonics, the timbre and sonorousness of the voice as well as the beginning and ending of the voice, the raising and lowering of the voice, and the tonic and expressive accents."

Hudgins[10] made use of the strobilion to correct the "fixed," *i.e.*, monotonous, voices of some deaf children, and the "wavering" voices of others. He made use of a neon lamp[11] flickering synchronously with the sound and illuminating a stroboscopic disc. Meyer[12] found that the neon lamp was less satisfactory than the acetylene flame, because the flame provided the light by which the child could read the sounds he was to speak, whereas the neon lamp would not glow until vocalization had commenced.

That the strobilion has not been more widely used in teaching correct pitch and inflection to the deaf is probably due to the practical difficulties involved in its application to the instruction of children:

1. It must be situated in a dark room, so that the child is not able to lip-read the instructor.

2. If a gas flame is used, a somewhat too loud or breathy sound will blow it out.

3. The fact that the gas cylinder frequently needs replacing is disadvantageous.

4. The mark on the disc is not easy for a child to follow or interpret.

5. No provision is made to cut out the harmonics which in the case of some vowels are considerably stronger than the fundamental. This leads to confusion; an overtone may be indicated at the very place where the teacher is endeavoring to have the child place the fundamental pitch level which is too low.

An instrument in which electric lamps are used to indicate pitch level was devised by Coyne.[13]

"The instrument indicates the comparative variations of different sounds by the lighting of lamps arranged in a vertical row at the front of the instrument. The person using it speaks or sings into a microphone. The sound vibrations of the voice are translated into electrical impulses, which are then amplified and applied to a series of electromagnetically-operated tuning forks. These in their turn are vibrated if the frequency of the voice agrees with their natural rate of vibration. Each tuning fork controls a lamp, but if several forks should be vibrating simultaneously, as often happens when a voice is rich in harmonics, only the lamp operated by the lowest of them will light."

Success was claimed for the instrument in the following educational procedures:

1. In producing voice.

2. In obtaining a sustained note without falling off or rising at the end.

3. In developing a sense of pitch.

4. In obtaining correct inflection in words and phrases.

Coyne uses 13 lights to cover the range of women's and children's voices, and a separate series of 13 for male voices.

III.–CHARACTERISTICS OF SPEECH SOUNDS.

Speech is generally recognized as being made up of vowels, "semivowels" (such as *l, m, n, r*), voiced consonants (such as *b, d, z*), and unvoiced consonants (such as *p, t, s*). Very little speech energy is carried by the latter; and, since they do not involve phonation, the "pitch" or fundamental tone of the voice does not affect them. The vowel sounds carry most of the speech energy. Inflection and, to a large extent, emphasis depend on the fundamental or lowest tone produced by the vibration of the vocal cords. The rise and fall of a sentence, the "upward inflection" at the end of a question, the differences in speech created by different emotions are all due to changes in the fundamentals of the spoken sounds. An instrument for indicating such changes should, therefore, respond only to the fundamental in the voice.

The main difficulty in constructing such an apparatus is the fact that only a small part of the speech energy is carried by the fundamental. As early as 1829, Willis[14] recognized that each vowel was characterized by a resonant note of particular pitch. Helmholtz[15] concluded that each vowel was characterized by fixed regions of resonance, which, like Willis, he thought were the same for men, women and children; but he found that, whereas certain vowels (*c*a*lm*, p*aw*, n*o*t) were due to single resonances, others (h*a*t, m*e*n, *ea*t) were due to double resonance, due to the doubly resonating cavities of the mouth and pharynx.

"The form of the oral cavity resembles a bottle with a narrow neck. The belly of the bottle is behind, in the pharynx, and its neck is the narrow passage between the upper surface of the tongue and the hard palate."[16]

In 1879, Bell[17] published the results of observations which led him to conclude that all vowels were due to double resonance.

Miller made an extensive study of the sounds of music and speech by the use of his phonodeik.[18] This is an instrument in which sound waves impinging on a diaphragm cause it to vibrate. These vibrations are communicated to a steel spindle carrying a small mirror, which reflects a spot of light onto a rotating drum. A wavy trace due to the sound can thus be recorded. Miller analyzed the wave form of many different

sounds by means of Fourier analysis. He concluded that some vowels[19] were characterized by a single fixed region of resonance, while some had two resonance peaks. The peaks occurred at the same frequencies, whatever type of voice was used to intone the vowels. Furthermore, only a very small percentage of the energy was expended on the fundamental tone.

In a more recent book, Miller[20] has stated his conception of the characteristics of vowel sounds as follows:

". . . each particular vowel is not characterized by a series of partials of fixed relative intensity, that is, it does not have a particular wave form; rather, it is characterized by a fixed region, or regions, of resonance or reinforcement. The greater part of the energy or intensity of the voice sound is in the partial or partials which fall within certain frequency limits, no matter at what pitch the vowel is uttered, or by what quality of voice. The variations in understandable speech and song are so great that we are accustomed to accept as the same vowel sounds which vary within fairly wide limits. . . .

"By far the greater part of the energy of the vibration (of the vowel *oo* as in gl*oo*m, intoned by a baritone voice on a pitch of 170 cycles) and of the loudness of the sound is contained in the second partial, there being just enough of the fundamental to give the impression of pitch; the other weaker partials combine to produce the tone quality in general of this particular voice. The second partial of frequency 340, having about 75 per cent of the energy of the sound, determines what vowel is uttered, gl*oo*m in this instance.

". . . Analysis (of the vowel *ee* as in b*ee*) shows that the fifteenth partial, . . ., which has a frequency of about 2,500 vibrations per second, contains a large part of the energy of the sound, 35 per cent, while the second partial is equally important. A direct comparison of the two records indicates that the vowel gl*oo*m is transformed into the vowel b*ee* by a very slight adjustment of the vowel cavities so as to add a new strong characteristic resonance of high frequency.

"Analysis shows that the vowel *a* as in f*a*ther is characterized by strong resonance, the frequency of which may vary from 900 to 1,100 vibrations per second. The record (of this

vowel sound intoned by a contralto voice) has a fundamental frequency of 487 cycles, and the second partial of frequency 974 contains 96 per cent of the energy of the sound.

"The curve (for vowel *o* as in n*o*, intoned at 226 cycles) has the greater part of its energy, 69 per cent in this instance, in the second partial having a frequency of 452. The pitch of the dominating partial, 452 vibrations per second, determines that this vowel is n*o*, in contrast with the vowel gl*oo*m . . ., the dominant partial of which has a frequency of 340. . . .

"An analysis of the vowel in the word m*a*t shows the presence of every partial from the fundamental to the twentieth overtone, inclusive; in another analysis of the same vowel, there are 18 partials, the highest being the twenty-fourth overtone."

Miller's results for the peak energies of the vowels may be summarized in Table I:

TABLE I.

Vowels — Class I (One Peak)	Peak at:
MA	910-1,050 cycles
MAW	732 cycles
MOW	461 cycles
MOO	326 cycles
Vowels — Class II (Two Peaks)	**Peaks at:**
MA	950 and 1,240 cycles
MAT	800 and 1,840 cycles
MET	691 and 1,840 cycles
MATE	488 and 2,461 cycles
MEET	308 and 3,100 cycles

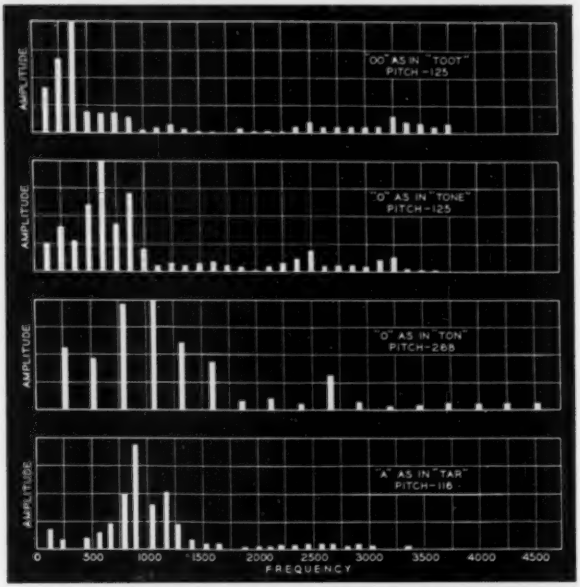
Paget made an interesting study on the vowel sounds.[21] He trained himself to recognize by ear the characteristic resonances in the vowel sounds. He observed double resonance peaks for all the vowel sounds. His results agreed well with those of Crandall and Sacia,[22] who investigated speech waves picked up by a condenser transmitter, amplified and recorded on phonograph records. The wave forms were analyzed with a harmonic analyzer. According to their findings, the frequencies characteristic of the different vowels bear a harmonic relation to the fundamental, so that a change in fundamental pitch should change the frequency at which the characteristic peak or peaks appear.

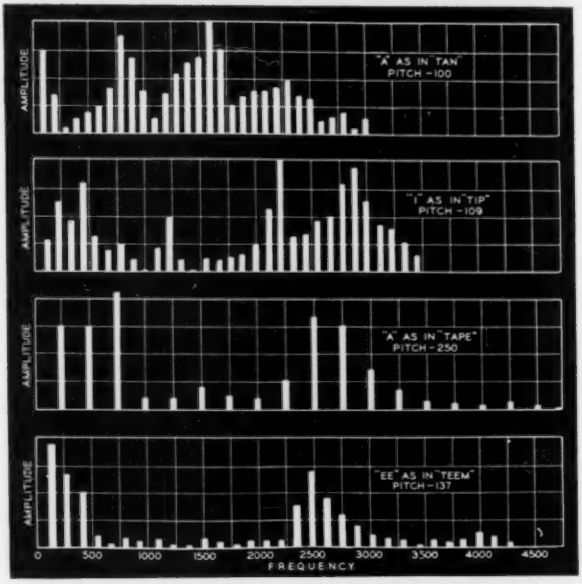
The two theories of vowel production are discussed by Fletcher:[23]

"The question arises as to what characteristics of a speech sound differentiate it from another speech sound. It is evident . . . that the pitch or the wave form of the vowel is not its distinguishing feature. Two theories of vowel production have been advanced; namely, the harmonic or steady state theory, and the inharmonic or transient theory. In spite of the fact that Helmholtz showed that these two theories were different only in the point of view and the method of representing the same mechanism of vowel production, we still have advocates of the two theories.

"The harmonic theory was first advocated by Wheatsone in 1837. According to this theory, the vocal cords generate a complex wave having a fundamental and a large number of harmonics. The component frequencies are all exact multiples of the fundamental. . . . When these waves pass through the throat, the mouth and the nasal cavities those frequencies near the resonant frequencies of these cavities are radiated into the air very much magnified, the amount depending upon the damping constant of the cavity. These reinforced frequency regions determine the vowel quality.

"According to the inharmonic theory of Willis (1829) and Herman, and now advocated by Scripture, the vocal cords act only as an agent for exciting the transient frequencies which are characteristic of the vocal cavities. A puff of air from the glottis sets the air in these cavities into vibration. This vibration soon diminishes until it is started anew by a second puff. According to this theory, the puffs do not necessarily follow each other periodically, and hence the name "inharmonic"; however, it is hard to see how the physical mechanism in the throat can produce anything but fairly regular puffs since these are controlled by the elastic properties of the vocal cords and the two resonant columns of air on either side of them. An examination of the records of speech sounds shows that this is true. The different waves succeed each other quite regularly. On the other hand, this examination also supports the view that these regular puffs do excite the transients of the mouth and throat cavities, for the amplitudes are large at the beginning of the wave and gradually die away toward the end. . . . When the pitch is high, the natural vibra-





Figs. 1 and 2. Acoustic spectra of various vowels.

tions do not have time to die down before another pulse sets them going again."

A diagram representing the "acoustic spectrum" of a vowel is a device which presents readily to the eye the characteristic frequencies. Fletcher[24] obtained such spectra from typical wave pictures taken with the high quality oscillograph. These spectra are reproduced in Figs. 1, 2, 3 and 4.

"Maxima . . . are plainly evident in these charts. Those spectra shown in Fig. 1 have one principal region of resonance, with indications of one or more regions of less importance, while those in Fig. 2 have two principal regions of resonance, with other smaller ones. It is well to emphasize here the fact that these charts represent the results obtained with typical voices. When the records of several speakers are analyzed, quite different acoustic spectra are obtained, but, in general, the regions of maximum amplitude are approximately the same.

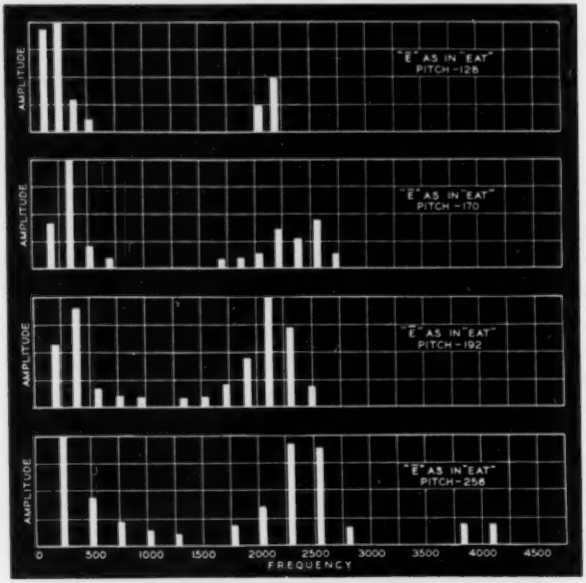
"In order to show the effect of pitch upon the acoustic spectra of vowel sounds, an analysis was made of vowels intoned at pitches corresponding to the notes of the chord; namely, at frequencies 128, 160, 192 and 256. The resulting spectra for ē and ā are shown in Figs. 3 and 4. It will be noticed that for the sound ē, the frequency regions 300 and 2,300 cycles, and for the sound ā, the regions 500 and 1,900 cycles are magnified."

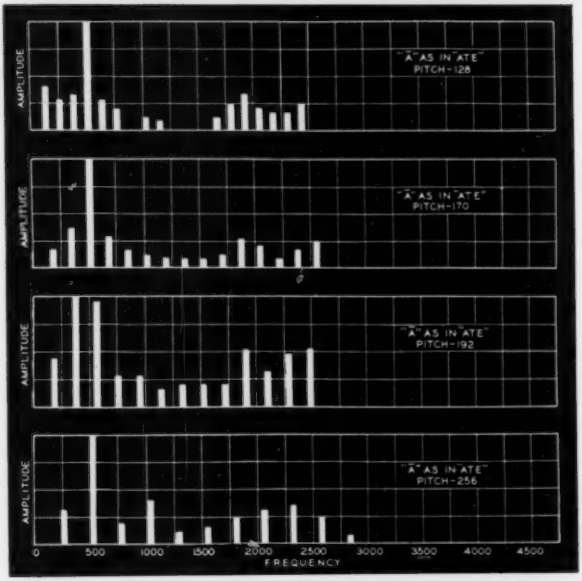
The same author constructed a table[25] giving the characteristic frequencies of the vowel sounds. The data for the table, reproduced in Table II, were taken from the work of Stumpf, Miller, Paget and Crandall.

TABLE II.

Characteristic Frequency of the Vowel Sounds.

Speech Sound	Low Frequency	High Frequency
u (pool)	400	800
u (put)	475	1,000
ō (tone)	500	850
a (talk)	600	950
o (ton)	700	1,150
a (father)	825	1,200
a (tap)	750	1,800
e (ten)	550	1,900
er (pert)	500	1,500
ā (tape)	550	2,100
i (tip)	450	2,200
ē (team)	375	2,400





Figs. 3 and 4. Changes in acoustic spectra with change in fundamentals.

The procedure of harmonic analysis of the wave traces made by speech sounds has been criticized by Scripture.[26] He showed how the product of a sine curve of the form $y = a \sin vx$ and the curve for logarithmic decrement, $y = e^{-x}$, would by Fourier analysis produce a constant plus a series of sine functions with pulsations in the ratios 1, 2, 3 — and with diminishing amplitudes. The result is correct mathematically, but incorrect as an indication of how the curve was derived. Scripture's own theory of vowel production is as follows:[27]

"Free vibration, such as in vowels, occurs as the movement of a vibrating system disturbed in its equilibrium and then left to itself. A vibration of this kind is produced when the air in the vocal cavity is rarefied or condensed by snapping the thumb out of the mouth, or by closing and suddenly opening the glottis. Such a sudden rarefaction or condensation may be termed a *puff*. Repeated puffs will produce a series of free vibrations."

Scripture gives examples of motion picture sound tracks of vowels: each track has a recognizable "profile," recognizable by the eye without analysis, and, analogously, the ear perceives each vowel also without analysis. His experiments in filtering showed that any frequency or range of frequency could be removed from the vowel vibrations without changing the vowel character.

His theory agrees with that of Willis, Helmholtz and Herman in regard to the *production* of vowels (*i.e.*, that a vowel consists of a series of waves or "contiguous vibration bits" that begin strong and diminish rapidly to zero, the vibrations being affected by strong factors of decrement); it differs in that Scripture does not consider perception to depend on any special frequency or groups of frequencies.

Recent workers in this field have, however, continued to use harmonic analysis in investigating the characteristics of vowels. An examination of various studies shows that the frequencies of peak resonance are not found to be fixed, as Miller's work[28] would lead one to suppose. Thus, Bartholomew[29] found that the frequencies of the low and high formants in various vowels differed in "good" and "bad" voices. Stein-

berg[30] analyzed the vowels in the sentence, "Joe took father's shoe bench out." He found slight variations in the positions of the frequency peaks with varying intensity and varying fundamental pitch. Lewis[31] analyzed oscillograms made by a trained baritone singing various vowels on different pitches. There was a change in the contour of the intensity-frequency curves at different pitches. Also, the relative intensity levels of the various partials changed as they changed in frequency, and there were changes of frequency with fundamental.

A study by Stout[32] of the vowels "ah," "oo" and "ee" indicated:

"*1.* That the most important change in the harmonic structure of the vowels 'ah,' 'oo' and 'ee' which accompanies an increase in intensity, pitch remaining constant, is an enhancement of the relative importance of the partials lying above the frequency 1,800 cycles; *2.* that this enhancement is greatest at the low pitch for the vowel 'ah,' greatest at the high pitch for the vowel 'ee' and about the same at all three pitches studied for the vowel 'oo.' *3.* That the increase in the fundamental which accompanies an increase in total intensity is very slight for the vowel 'ah,' considerably more for the vowel 'oo' and greatest for the vowel 'ee.'

"The results of the study also indicate: *1.* That the most important change in the harmonic structure of the vowels 'ah,' 'oo' and 'ee' that occurs with a rise in pitch, intensity remaining constant, is a decrease in the relative intensity of the partials lying above the frequency 1,800 cycles . . ; *2.* that in the case of all three vowels, the fundamental appears to absorb a considerable part of the energy which has shifted from the high frequency regions to the low.

"That there is no consistent change in frequency location of the major intensity areas with either an increase in total intensity or a rise in pitch is quite definitely indicated by the results of this study."

Black[33] states that—

"the physical pattern of a vowel varies *1.* with frequency; *2.* with intensity; *3.* within itself from wave to wave — a polypthongal state; *4.* between different speakers; *5.* within the speech of the same speaker when the vowel is bounded by different consonants."

IV.–CONSTRUCTION OF THE INFLECTOR.[34]

Whatever theory of vowel production be accepted, it is clear that the partials would be troublesome in any instrument intended to respond to the fundamental only. A mechanically resonating system would respond to harmonics as well as to the fundamental, which would make it impossible to use for the purpose intended, *i.e.*, the instruction of deaf children in the correct pitch and modulation of the voice. A preliminary investigation with tuned oscillating circuits showed that extremely complex problems would be involved.

It was thought, then, that an electronic frequency metre of the type described by Hunt[35] could be adapted for use with

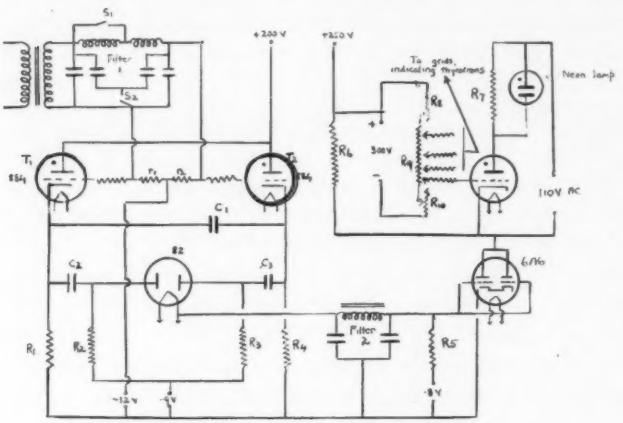


Fig. 5. Circuit for the inflection indicator.

deaf children. Hunt himself suggested that such an adaptation might be made. Fig. 5 presents schematically a circuit which was found to be suitable, and which is now embodied in an instrument in use at Central Institute for the Deaf.

The frequency metre includes thyratrons T_1 and T_2, condensers C_1, C_2, C_3, resistances R_1, R_2, R_3, R_4 and the type 82 rectifier. The operation of this part of the circuit is as follows:

If T_1 be conducting, and T_2 nonconducting, then condensers C_1 and C_2 are charged up by an amount equal to the voltage drop across R_1, condenser C_3 being uncharged. If now a

signal from the amplifier be applied through the resistance in the grid circuit C_2 in such a way that the grid of T_2 becomes sufficiently positive for T_2 to fire, the potential of the cathode of T_2 will be raised abruptly, the voltage drop across R_4 being equal to the difference between the plate supply and the tube drop in T_2. Since the voltage across C_1 cannot alter instantaneously, the cathode of T_1 will for a very short time become positive with respect to its plate. Since the grid of T_1 is at the same time negative, the arc in T_1 will be extinguished, provided that the deionization time of the thyratron is not greater than the time required for C_1 to discharge through R_1. Similarly, since C_2 and C_3 cannot alter instantaneously, the plates of the type 82 rectifier tube will be raised alternately to a positive voltage equal to the voltage drop across R_2 or R_3 and current impulses will be delivered to the next part of the circuit.

The average current delivered will be proportional to the number of impulses delivered per second. It is, therefore, proportional to the frequency with which the polarity of the input voltage changes its sign. Thus, current varies with the frequency of the fundamental, provided that no harmonic in the sound is loud enough to cause the voltage to reverse more than twice during each fundamental period. This frequency metre, therefore, provides current which is independent of the wave form of the signal, and which increases with increasing frequency of the fundamental.

The current delivered by the rectifier tube is smoothed by filter 2, which consists of a 10-henry choke coil and two 2-microfarad condensers. The voltage drop across R_5 produced by the rectified current is applied to the grids of the 6A6 double-triode which was used because of its low plate resistance (11,000 ohms) and its amplification factor of 35. Most of the voltage drop in the plate circuit of the double-triode appears across the high resistance R_6 (100,000 ohms).

The indicating part of the circuit consists of seven thyratrons, with a three watt neon glow lamp across a 6,000 ohm resistor in the plate circuit of each. The grid bias for each thyratron is tapped off from a bleeder-resistance carrying direct current. The cathodes are connected to the plates of the 6A6, and the positive end of R_6 is connected with the positive

end of the bleeder. An indicating thyratron will then flash if the voltage drop across R_6 carries the grid sufficiently positive. The thyratrons here used (type RCA 884) will flash when the negative grid bias is less than, roughly, one-tenth of the plate voltage. If the voltage drop across R_6 becomes Ef due to frequency f of the input signal, then those thyratrons will flash whose grid bias is less than Ef plus one-tenth of the plate voltage. It is possible, then, to arrange the grid bias of the tubes so that they flash at any desired frequencies. When the discharge in the thyratron has commenced, the voltage across R_7 (6,000 ohms) is equal to the plate supply (110 volts) less the drop in the tube (about 15 volts). This is sufficient to cause the near lamp to glow. The seven neon lamps are arranged in a vertical row, the grid bias of the controlling thyratrons increasing from the lowest to the highest. A sound of increasing frequency will then cause successively higher lamps to glow. The top lamp to glow will indicate the frequency of the input signal.

A crystal microphone and amplifier are used to supply the input voltage for the frequency metre. The amplifier in this instrument is a Thordarson eight watt model. A Thordarson dual tone control is used with the amplifier. The controls are left in the position which will give maximum bass output, so that the fundmental is hereby somewhat strengthened. A low pass filter is also necessary to remove frequencies higher than the highest to be dealt with by the instrument. Filter 1 is the low pass filter. The instrument is intended for use with all types of voices, and it is advantageous to make the filter cut off at as low a frequency as possible. It is, therefore, so arranged that three different valves of cut-off frequency can be attained, for low, middle and high voices, respectively. The filter consists of two inductance coils, of 0.133 and 0.150 henries. The latter can be shorted out by closing switch S_1. The four condensers in filter 1 have a capacitance of 1.29 microfarads each. With switch S_2 open, only two of these are brought into the circuit. The highest cut-off frequency is obtained with S_1 closed and S_2 open. Cut-off frequency is then given by:

$$f = \frac{1}{\pi\sqrt{LC}} = \frac{1}{\pi\sqrt{.133 \times 2.58 \times 10^{-6}}}$$

$$= \text{about } 549 \text{ cycles}$$

Cut-off frequency is obtained for "middle" voices with S_1 and S_2 open.

Cut-off frequency is given by

$$f = \frac{1}{\pi\sqrt{LC}} = \frac{1}{\pi\sqrt{.283 \times 2.58 \times 10^{-6}}}$$
$$= \text{about } 370 \text{ cycles}$$

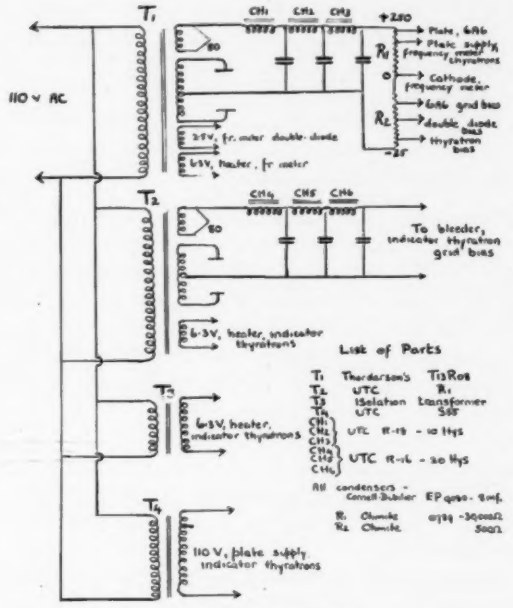


Fig. 6. Power supply.

Cut-off frequency is obtained for low voices by bringing the two extra condensers into the circuit; *i.e.*, by closing switch S_2, S_1 remaining open. Cut-off frequency is then given by

$$f = \frac{1}{\pi\sqrt{.282 \times 5.16 \times 10^{-6}}}$$
$$= \text{about } 260 \text{ cycles}$$

A power supply is embodied in the instrument, so that it can be plugged into the 110 A.C. supply. The schematic diagram is shown in Fig. 6.

Transformer T_1 has four secondary windings. The five volt, three amp. secondary supplies the rectifier tube (type RCA 80). The 2.5 volt, six amp. centre tapped winding supplies filament current for the rectifier tube (type RCA 82) of the frequency metre. A 6.3 volt, 3.3 amp. winding heats the filaments of the frequency metre thyratrons and the 6A6. The high voltage (700 volts, centre-tapped) is rectified and smoothed in the usual way. The 300 volts which appear across the bleeder resistance is sufficient to supply the plate voltage and bias of the frequency metre tubes. The bleeder is in two parts (see Fig. 6): R_1, 30,000 ohms, and R_2, 500 ohms; the various voltages are tapped off as shown. R_2 is on the same chassis as the frequency metre, so that the correct critical adjustments of the bias for the thyratrons, rectifier tube and 6A6 may conveniently be made.

Transformer T_2 has three secondary windings. The five volt, two amp. secondary supplies the rectifying tube (type RCA 80) for the bias supply of the indicating thyratrons. The 6.3 volt, two amp. winding supplies heater current for three of the indicating thyratrons. The high voltage is rectified and smoothed, and leads are brought from the power supply chassis to the bleeder resistance located near the indicating thyratrons. This bleeder resistance is in three parts, indicated in Fig. 5 as R_8, R_9, R_{10}. R_9 is a 10 inch, 15,000 ohm variable resistor. The bias of the seven indicating thyratrons is tapped off at various points along R_9. R_8 and R_{10} are variable 5,000 ohm resistors. By varying one or both of these, the grid bias applied to the indicating thyratrons is altered, so that the range of frequencies to which the lamps respond is altered. Thus, by shorting out both R_8 and R_{10} the range covered is from 90 to 540 cycles. With R_8 and R_{10} both fully in circuit, the range covered is about an octave in the usual speech range. Thus, the seven lamps may be made to cover a large or small range in low, middle or high voices. The usefulness of this feature will be discussed in a later section.

Transformer T_3 has a single secondary winding, supplying 6.3 volts at four amps. to four of the indicating thyratrons. T_4 is an isolating transformer which supplies 110 volts to the plates of the indicating thyratrons. The plate supply must be A.C. so that the grid may regain control after a tube has fired.

Various details have been incorporated in the instrument

to add to its value in instructing deaf children. So that they may feel the vibrations due to a sound while they are watching the lights, a permanent magnet speaker is included; this is connected across the four ohm output terminals of the amplifier. There is ample power for its operation, as the input to the frequency metre, working off the 500 ohm output terminals of the amplifier is connected across two resistors (see Fig. 5: r_1 and r_2) of 1,500 ohms each.

Coyne[36] has been followed in the arrangement of the lights in a vertical row, with a slate next to them on which the teacher may write whatever is necessary. An output metre is included to indicate the intensity of the voice.

V.–THE INFLECTOR IN USE.

A.— Observations on the Vowel Sounds:

The instrument described in the last section has been found to answer satisfactorily to its purpose; namely, that of responding only to the fundamental of the voice. The following three features are active in insuring such a response:

1. The principle of the frequency metre, which in a complex sound responds only to the fundamental, as long as it is stronger than any of the harmonics.

2. The Thordarson dual tone control, left at a setting in which the amplifier output is distorted to give maximum gain to the lowest tones.

3. The low pass filter, used to give as low a cut-off frequency as is convenient.

There are still some speech sounds in which the instrument responds to the overtones rather than to the fundamental. If the various controls are correctly used, these need not appear, so that they are not a disturbing factor while the instrument is being used for instructing deaf children. It is possible, however, by noting the circumstances in which these overtones appear, to make certain observations of a qualitative nature on the characteristics of vowel sounds, although the inflector was not intended for analytical purposes.

1. Some overtones appear when certain vowel sounds are intoned, but they are not invariably present. They affect the

instrument when the vowels are intoned at some definite pitch and with some particular intensity. Since they will appear only when they are considerably stronger than the fundamental, it can be seen that they imply resonance "peaks." These peaks are, therefore, not fixed for all types of voice and all fundamental frequencies, as is suggested by the work of Miller.[37]

2. Overtones due to vowels *ah* (as in f*a*ther) and *aw* (as in p*aw*) are observed when the cut-off frequency is about 550 cycles per second. It would seem, therefore, that these vowels possess strong resonance peaks between the fundamental pitch and 550 cycles. It is interesting to note the position of the lowest resonance peaks as given by various workers for vowels *ah* and *aw*.

TABLE III.

Author	Lowest Res. Peak, "ah"	Lowest Res. Peak, "aw"
Miller[37]	ca. 910 c.p.s.	732 c.p.s.
Fletcher[38]	ca. 825 c.p.s.	600 c.p.s.
Paget[39]	ca. 730 c.p.s.	512-574 c.p.s.
Crandall and Sacia[40]	ca. 500 c.p.s.	ca. 650 c.p.s.

The vowel *ah* intoned at various intensities and with various fundamental frequencies was one of the three investigated by Stout.[41] His results were presented in the form of acoustic spectra, definite regions for the lowest characteristic frequency not being given. The spectra for this vowel show some shifting of the relative intensities of the harmonics in varying conditions of intonation. He concluded that with rising intensity there was a slight increase in the relative intensity of the fundamental; and a more marked rise in the relative intensity of the fundamental with rising pitch. Observations with the inflection indicator show, however, that the fundamental is relatively stronger in vowel *sh* with small intensities and with relatively low pitch. When the vowel is intoned by a female voice, the strong partial or partials below 550 cycles appear as the intensity of the voice is increased, and as the pitch is raised. The harmonics appearing in both the vowels *ah* and *aw* disappear when the cut-off frequency is 370 cycles per second. This value of cut-off frequency is sufficiently high for almost all women and children's speech, so that, as stated, the presence of the harmonics due to these

vowels need not be a disturbing factor in using the instrument for purposes of instruction.

3. Bartholomew's[42] finding that the presence and relative intensity of the "low formant" depend on the quality of voice used is borne out by observations with the inflector. In general, it would appear that the "clearer" the voice, the freer it is from harmonics below 550 cycles per second. Some of the vowels when intoned by hearing persons will show no evidence of harmonics in this region, or at any rate of harmonics sufficiently strong to be indicated by the instrument; yet when intoned by deaf children with noticeably poor voice quality, harmonics will be evidenced.

4. Vowels *oo* and *ee* have been represented by most workers as having resonance peaks in the range 300-400 cycles. It was expected, therefore, that they would evidence disturbing harmonics. It would appear, however, that these resonance peaks are not sufficiently strong to show up on the inflection indicator. The vowel *ee* is particularly free from evidence of harmonics with most voices.

5. In general, adult male voices show the most evidence of strong harmonics in the low frequency region.

6. Vowels which in some circumstances give evidence of strong harmonics can with practice be so intoned that the harmonics do not appear. This is true except with the vowel *aw*. Thus, it would seem that the overtones in the voice in this low range are to some extent subject to conscious control, while their presence or absence does not affect the intelligibility of the vowel. Generally the clearer, "sweeter" voice is the more free from harmonics in the region below 550 cycles.

The above observations suggest that the instrument might be used as a means of determining very readily some of the characteristics of speech sounds; the usual methods require rather laborious analysis. Interesting comparisons might be made between the characteristics of the speech of deaf and of hearing persons. Variations in the relative strength of the low partials with varying pitch and intensity could systematically be studied. The usefulness of the instrument for analytical purposes would be enhanced by making the low pass filter continuously variable, and by setting the dual tone control for undistorted output. The frequency of each harmonic

stronger than the fundamental could then be determined for any speech sound. The instrument would register the strongest harmonic occurring below whatever value of cut-off frequency was used. The acoustic spectra of speech sounds could thus be mapped out, at any rate for the low frequency region. The region of frequencies to which the instrument would respond could be extended by changing the constants of the frequency metre.

B.— Use of the Inflector in Instructing the Deaf:

It has been found that correct inflection in words and phrases cannot be taught to a child until he is able to place any speech sound at any desired pitch. There are certain vowels, notably *ee* (as in b*ee*) and *oo* (as in y*ou*), which deaf children tend to phonate at a high pitch; whereas others, notably *aw* (as in p*aw*), are usually phonated at a low pitch. Considerable practice is necessary so that any vowel or voiced consonant may be given at any desired pitch. The indicator is particularly useful for this type of practice. The child is shown how to keep a certain number of lamps glowing steadily while phonating a sound; he then practices until he is able to do the same. It has been found helpful with many children to start pitch exercises with the sound *m*. Phonation of this sound requires simply the placing of the lips together while giving voice. The children, guided partly by the tactile sensations obtained from putting their fingers on the loudspeaker, are usually able to hit the desired pitch after a certain amount of trial. After some work to develop kinesthetic memory for the pitch that causes any lamp to glow, practice is given in phonating all the vowels at specific pitch levels. With some children it may be necessary to work initially for broad pitch change. The pitch range covered by the lamps may be increased for this purpose by turning the knobs which short out parts of resistance R_8 and R_{10} (see Fig. 5). The majority of the children at Central Institute for the Deaf[43, 44] have learned how to change the pitch of their voices over a wide interval, and the inflector is useful in showing them how to change the pitch of their voices within the rather narrow range used in normal speech. For this sort of work, resistances R_8 and R_{10} are so adjusted that the frequencies at which the lamps glow are about one tone apart. The natural pitch level of the voice varies considerably in different children. It

is a good plan for the teacher to set the range controls for each pupil so that the medium voice pitch lights the middle lamp of the column. Care should be taken to have the switch which controls the low pass filter in the appropriate position. The teacher should be on the alert to change the position of the switch whenever necessary.

It may be noted that the best results are obtained from the children when they are permitted to think only of pitch; they should not be distracted by having their attention drawn to faults in voice quality, volume or articulation. Connery[45] has

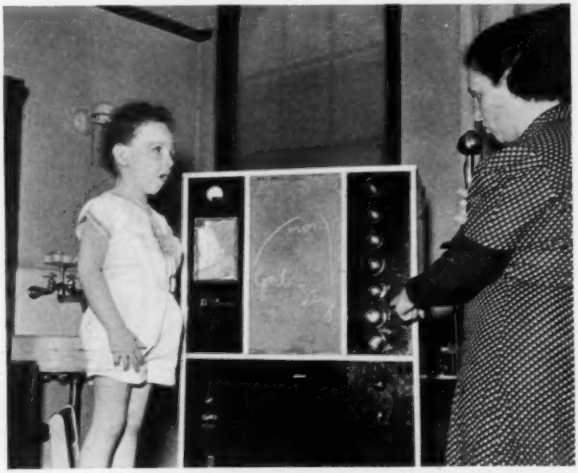


Fig. 7.

pointed out the necessity for having articulation perfect in any sound or sequence of sounds before pitch work is commenced. With simple sounds, vowels or semivowels, the teacher may work for better volume when the child is able to phonate at the desired pitch. In some cases, this can be achieved by a sort of trick; the gain in the amplifier may be turned down slightly so that a good resonant tone is required to make the lamps glow steadily. Some children attempt to get variations in the height of the light column by varying the volume of the sound, usually employing soft voice in the attempt to get low pitch. Since low pitch is often more diffi-

cult for the child to produce, it is advisable in such a case to have the gain control of the amplifier turned up to its maximum. The child can then see that a high tone produces the same effect on the lamps, whether it is soft or loud.

VI.—RESULTS AND CONCLUSIONS.

Considerable time will have to elapse before the value of the inflector can be accurately estimated. From the work already done, however, it would appear that the instrument should be exceedingly helpful. Children of various ages have had short periods of work on the indicator, and without exception they have displayed considerable interest. Most of them are eager to work by themselves at achieving what is required. The motivation is much better than where the child is completely dependent on the teacher's work or look of approval. Successful achievement of a required pitch level can be followed by practice on the same sound, without further prompting by the teacher. Some children have for the first time formed the conception of holding a note steadily without allowing it to fall or rise in pitch. It may be suggested that when once a child is able to place the vowels at various pitch levels, he need then only be told that an emphasized syllable or word is phonated at a slightly higher pitch level than the rest of the speech sounds accompanying it. This was well demonstrated by "A," who is a well educated young man. "A's" speech is fluent and idiomatic, but his voice is usually a falsetto, and he enunciates the high vowels "oo" and "ee," and sometimes other vowels at a high pitch level. Some words are unintelligible because of the lack of correct inflection. "A" had apparently had no previous work in pitch change or inflection. After working with the inflector for two short periods, separated by a week, he was able to lower his voice at will, and to give correct inflection to various words when he was told which syllable to accent, and also that the accented syllable is always slightly higher. At first he claimed that he could not feel the differences in sensation produced when he intoned a sound steadily, or allowed it to fall by a tone. After observing the behavior of the lights, he was able to produce either a steady or an unsteady tone voluntarily, and he could feel the difference in kinesthetic sensation. It is probable, however, that considerable work will have to be done to overcome bad speech habits which have become set.

It seems advisable that quite young deaf children be trained with the inflection indicator so that they may learn as early as possible to inflect their voices within the narrow range used in ordinary speech. It would probably be advantageous to break the habit of giving certain vowels at certain pitch levels as soon as the child is old enough to be able to work with the instrument. Children of 6 and 7 years of age have shown that they are capable of deriving considerable benefit. J. F., a boy age 6 years, was able to give *ee* at four different intervals separated by a tone, after about 10 minutes' work. It would naturally be very difficult for a teacher to attain the same results without a visual symbol of what was required, and visual evidence of the success or failure of his efforts. It is recommended that children as young as this should be allowed to experiment with the inflector until they themselves form the association between the kinesthetic sensations accompanying pitch change and the corresponding behavior of the lamps. The learning problem involved would seem best solved by the method of trial and error.

Acknowledgments: We wish to express our gratitude to Dr. Max A. Goldstein, of Central Institute for the Deaf, and to Prof. Roy Glasgow, of Washington University, St. Louis, for their kind help and constant encouragement.

BIBLIOGRAPHY.

BARTHOLOMEW, W.: A Physical Definition of Good Voice Quality. *Jour. Acous. Soc. Amer.*, 6:25, July, 1934.

BELL, ALEXANDER G.: Vowel Theories. *Amer. Jour. Otol.*, 1:257, July, 1879.

BLACK, JOHN W.: The Effect of the Consonant on the Vowel. *Jour. Acous. Soc. Amer.*, 10:203, Jan., 1939.

CONNERY, JULIA M., and YOUNG, IRENE: Voice Building. Copyright, 1935, by Julia M. Connery.

COYNE, A. E.: The Coyne Voice Pitch Indicator. *Volta Rev.*, 40:437-439, 468-469, Aug., 1938.

COYNE, A. E.: More About the Voice Pitch Indicator. *Volta Rev.*, 40-549-552, Oct., 1938.

CRANDALL, I. B., and SACIA, C. T.: A Dynamical Study of the Vowel Sounds. *Bell Sys. Tech. Jour.*, 3:232-237, 1924.

FERRERI, GUILIO: The Audiphone-Stroboscope. *Volta Rev.*, 36:135, 1934.

FLETCHER, HARVEY: Speech and Hearing. D. Van Nostrand Co., New York, 1929.

GAULT, H. H.: Experiments on Tactual Interpretation of Oral Speech. *Jour. Abnorm. and Soc. Psychol.*, 19:155-159, 1924.

GOLDSTEIN, MAX A.: Problems of the Deaf. THE LARYNGOSCOPE PRESS, St. Louis, 1933.

GOLDSTEIN, MAX A.: The Acoustic Method. THE LARYNGOSCOPE PRESS, St. Louis, 1939.

HELMHOLTZ, H. VON: Sensations of Tone. Longmans, Green and Co., New York, 1912.

HUDGENS, CLARENCE V.: Visual Aids in the Correction of Speech. *Volta Rev.*, 37:637, Nov., 1935.

HUNT, FREDERICK V.: A Direct Reading Frequency Metre Suitable for High Speed Recording. *Rev. Sci. Instr.*, Vol. 6, Feb., 1935.

LEWIS, DON: Vocal Resonance. *Jour. Acous. Soc. Amer.*, 8:91-99, Oct., 1936.

MEYER, MAX F.: Fitting Into a Silent World. Univ. Mo. Studies, 9:2, 1934.

MILLER, DAYTON C.: Science of Musical Sounds. Macmillan Co., New York, 1922.

MILLER, DAYTON C.: Sound Waves, Their Shape and Speed. Macmillan Co., New York, 1937.

PAGET, SIR RICHARD: Human Speech. Harcourt Brace and Co., New York, 1930.

RHODES, F. L.: Beginnings of Telephony. Harper's, New York, 1929.

SCRIPTURE, E. W.: Stuttering, Lisping and Correction of Speech of the Deaf. Macmillan Co., New York, 1923.

SCRIPTURE, E. W.: Failure of Fourier Analysis Applied to Vowel Vibrations. *Nature*, 136:223, Aug., 1935.

SCRIPTURE, E. W.: Puff and Profile Theory of the Vowels. *Nature*, 136:435, Nov., 1935.

STEINBERG, JOHN C.: Application of Sound Measuring Instruments to the Study of Phonetic Problems. *Jour. Acous. Soc. Amer.*, 6:16, July, 1934.

STOUT, BARRETT: Harmonic Structure of Vowels in Singing in Relation to Pitch and Intensity. *Jour. Acous. Soc. Amer.*, 10:137-146, Oct., 1938.

WILLIS, R. G.: *Trans. Cambridge Philosoph. Soc.*, 3:231, 1829.

REFERENCES.

1. GAULT, R. H.: Experiments on Tactual Interpretation of Oral Speech. *Jour. Abnorm. and Soc. Psychol.*, 19:155-159, 1924.

2. RHODES, F. L.: Beginnings of Telephony, p. 8. Harper's, New York, 1929.

3. SCRIPTURE, E. W.: Stuttering, Lisping and Correction of the Speech of the Deaf, pp. 244-245. 2nd Ed. Macmillan Co., New York, 1923.

4. *Ibid.*, pp. 246-248.

5. *Ibid.*, p. 252.

6. GOLDSTEIN, MAX A.: Problems of the Deaf, p. 140. THE LARYNGOSCOPE, PRESS, St. Louis, 1933.

7. MEYER, MAX F.: Fitting Into a Silent World. Univ. of Mo. Studies, 9:2:83-90, 1934.

8. *Ibid.*, pp. 39-43.

9. FERRERI, GUILIO: The Audiphone-Stroboscope. *Volta Rev.*, 36:135, March, 1934.

10. HUDGINS, CLARENCE V.: Visual Aids in the Correction of Speech. *Volta Rev.*, 37:637, Nov., 1935.

11. SEASHORE, C. E.: Some New Instruments in the Iowa Laboratory for the Psychology of Music. *Jour. Acous. Soc. Amer.*, 2:75-77, July, 1930.

12. Op. cit., p. 87.

13. COYNE: The Coyne Voice Pitch Indicator. *Volta Rev.*, 40:437-439, 468-469, Aug., 1938.

14. WILLIS: *Trans. Cambridge Philosoph. Soc.*, 3:231, 1829.

15. HELMHOLTZ, H. VON: Sensations of Tone, Translated, Alexander J. Ellis, p. 103. Longmans, Green and Co., New York, 1912.

16. HELMHOLTZ, H. VON: *Ibid.*, p. 107.

17. BELL, ALEXANDER GRAHAM: Vowel Theories. *Amer. Jour. Otol.*, Vol. I. July, 1879.

18. MILLER, DAYTON C.: Science of Musical Sounds, p. 77 *et seq.* 2nd Ed. Macmillan Co., New York, 1922.

19. *Ibid.*, p. 215.

20. MILLER, DAYTON C.: Sound Waves, Their Shape and Speed, p. 58 *et seq.* Macmillan Co., New York, 1937.

21. PAGET, SIR RICHARD: Human Speech, p. 40 *et seq.* Harcourt Brace and Co., New York, 1930.

22. CRANDALL, I. B., and SACIA, C. F.: A Dynamical Study of the Vowel Sounds. *Bell Sys. Tech. Jour.*, 3:232-237 1924.

23. FLETCHER, HARVEY: Speech and Hearing, p. 46 *et seq.* D. Van Nostrand Co., New York, 1929.

24. Op. cit., p. 51.

25. Op. cit., p. 58.

26. SCRIPTURE, E. W.: Failure of Fourier Analysis Applied to Vowel Vibrations. *Nature*, 136:223, Aug., 1935.

27. SCRIPTURE, E. W.: Puff and Profile Theory of the Vowels. *Nature*, 136:435, Nov., 1935.

28. Loc. cit.

29. BARTHOLOMEW: A Physical Definition of "Good Voice Quality." *Jour. Acous. Soc. Amer.*, 6:25, July, 1934.

30. STEINBERG, JOHN C.: Application of Sound Measuring Instruments to the Study of Phonetic Problems. *Jour. Acous. Soc. Amer.*, 6:16, July, 1934.

31. LEWIS, DON: Vocal Resonance. *Jour. Acous. Soc. Amer.*, 8:91-99, Oct., 1936.

32. STOUT, BARRETT: The Harmonic Structure of Vowels in Singing in Relation to Pitch and Intensity. *Jour. Acous. Soc. Amer.*, 10:137-146, Oct., 1938.

33. BLACK, JOHN W.: The Effect of the Consonant on the Vowel. *Jour. Acous. Soc. Amer.*, 10:203, Jan., 1939.

34. The instrument was developed at the Radio Engineering Department of Washington University, in collaboration with Mr. H. J. Zimmerman, whose assistance is gratefully acknowledged.

35. HUNT, FREDERICK V.: A Direct Reading Frequency Metre Suitable for High Speed Recording. *Rev. Scien. Instr.*, 6:43:47, Feb., 1935.

36. COYNE, A. E.: Loc. cit.

37. MILLER, DAYTON C.: Loc. cit.

38. FLETCHER, HARVEY: Loc. cit.

39. PAGET, SIR RICHARD: Op. cit., p. 42.

40. CRANDALL, I. B., and SACIA, C. T.: Loc. cit.

41. STOUT, BARRETT: Op. cit., p. 139.

42. BARTHOLOMEW: Op. cit.

43. GOLDSTEIN, MAX A.: The Acoustic Method, pp. 46-47. THE LARYNGOSCOPE PRESS, St. Louis, 1939.

44. CONNERY, JULIA, and YOUNG, IRENE: Voice Building, p. 18 *et seq.*, p. 31 *et seq.* Copyright 1935, Julia M. Connery.

45. Op. cit., p. 22.

MISSISSIPPI VALLEY MEDICAL SOCIETY MEETS AT BURLINGTON, IOWA, SEPT. 27-28-29.

The Fifth Annual Meeting of the Mississippi Valley Medical Society will be held in the new $500,000 Municipal Auditorium at Burlington, Iowa, Sept. 27, 28 and 29. There will be 32 clinicians on the program who will give 50 lectures, clinical demonstrations, etc. An all-St. Louis program, with 14 clinical teachers from St. Louis University and Washington University, will feature the first day. Two short courses of instruction of four hours each will be given in internal medicine and gynecology by Dr. Fred H. Smith, Head of the Department of Medicine, and Dr. E. D. Plass, Head of the Department of Obstetrics and Gynecology, University of Iowa. Among the speakers will be Dr. Rock Sleyster, President of the American Medical Association; Dr. Evarts A. Graham and Dr. V. P. Blair, Professors of Surgery, Washington University; Dr. W. T. Coughlin, Professor of Surgery, and Dr. Charles H. Neilson, Professor of Medicine, St. Louis University; Dr. Frederick F. Boyce, Assistant Professor of Surgery, Louisiana University; Dr. Arthur E. Hertzler, Professor of Surgery, University of Kansas; Dr. Karl Goldhamer, formerly Roentgenologist, University of Vienna. There will be a big technical and scientific exhibit hall. A complimentary stag supper will be given on Sept. 27, and banquet on Sept. 28. Every ethical physician is cordially invited to attend. Detailed program may be secured from Harold Swanberg, M.D., Secretary, M.V.M.S., 209-224 W.C.U. building, Quincy, Ill.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

Meeting of April 24, 1939.

Osteoma of the Maxillary Sinus. Dr. Eugene Orr.

J. C. D., age 65 years, male, consulted me on March 16, 1939, on account of failing vision. There was a diffuse though quiet retinochoroiditis, both eyes. He was known to be a controlled diabetic. At the time seen, the urinalysis was negative, and blood sugar was 150; Wassermann negative; blood pressure 150/90; he was edentulous and tonsilless. X-ray of sinuses showed all sinuses clear except left antrum. Left antrum showed cloudy, and springing from the posterior aspect of the antrum was a dense shadow. This shadow was apparently within the antrum. The antrum was irrigated through the inferior meatus. There was a moderate amount of pus. X-ray was made with the cannula in situ. Luc-Caldwell operation under local anesthesia was done, March 20, and polypoid membrane removed. The osteoma was attached to the extreme posterior and somewhat lateral aspect of the infraorbital plate. Its size, roughly estimated, varied in diameter from slightly less than an inch to a little more than one-half inch. It was attached by a very short pedicle, slightly less than one-half inch in diameter. It was slightly nodular, and macroscopically, at least, was not covered by any sort of membrane. It was extremely hard; in fact, one could barely make an impression on it with gouge and mallet. We were unable to remove it with instruments at hand. As there was no evidence of orbital invasion, our future policy will be to observe it radiographically from time to time and if there is any tendency to invade the orbit or increase in size, further effort at its removal will be made.